AMERICAN JOURNAL

OF

OPHTHALMOLOGY

SERIES 3 VOLUME 8

1925

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PUBLISHED MONTHLY BY THE OPHTHALMIC PUBLISHING COMPANY
7 West Madison Street, Chicago, Illinois.

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American Journal of Ophthalmology

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Human Fetus 97 mm. g. l. Stain the Van Geisen.

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Figure 2. Cross section of orbital portion of optic nerve of fellow eye. Objective 16 mm., Ocular 10. (d) Dural sheath. (p) Pial sheath. (g) Glial septa. (f) Fibrous tissue arising from pial sheath and growing into nerve along the glial septa.

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(f) Fibrous septa. (gl) Glial cells forming anlage of lamina. (f^1) Bundle of fibrous connective tissue growing from the sclera into posterior layers of anlage of lamina. (scl) Sclera.

Figure 2. Cross section of orbital portion of optic nerve of fellow eye of same fetus. Obj. 16 mm. Oc. 10. (d) Dural sheath. (p) Pial sheath. (f) Fibrous connective tissue growing from pial sheath into nerve to form septa. (h) Central vessels.

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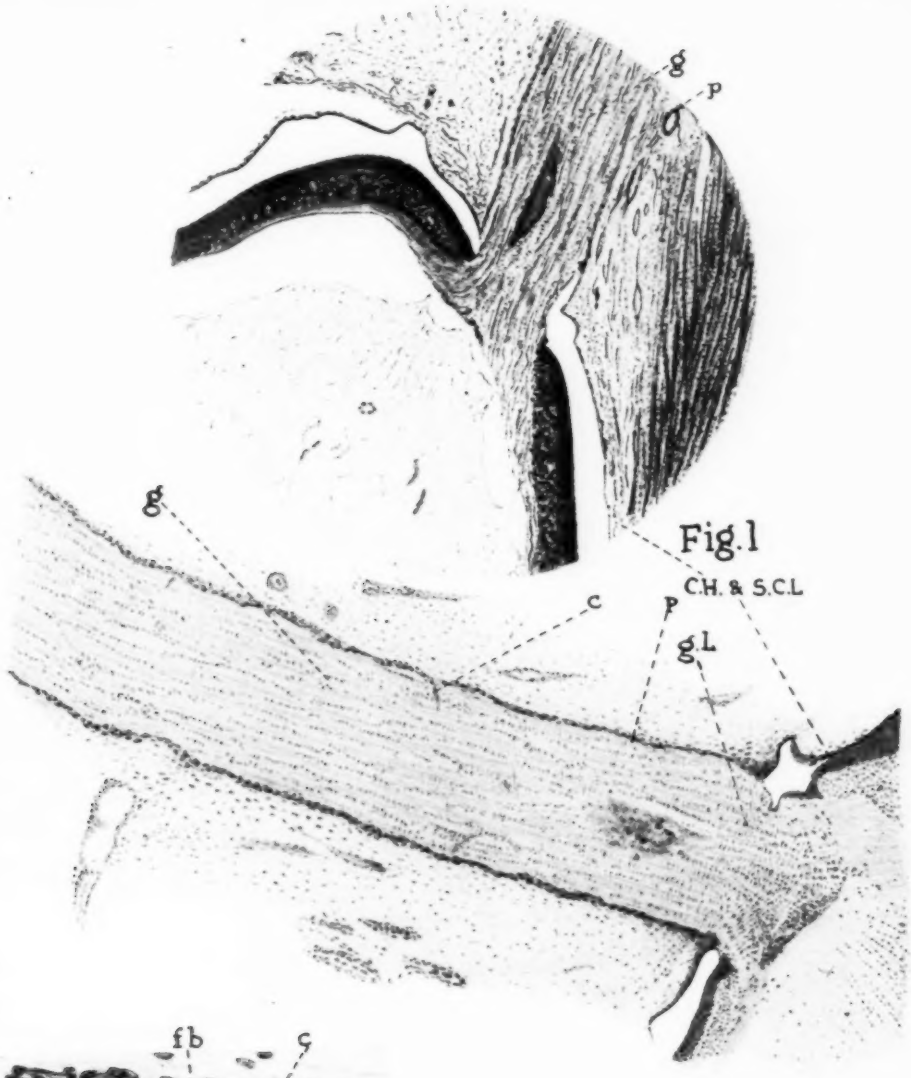

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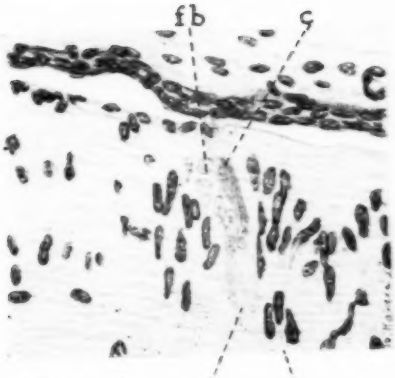

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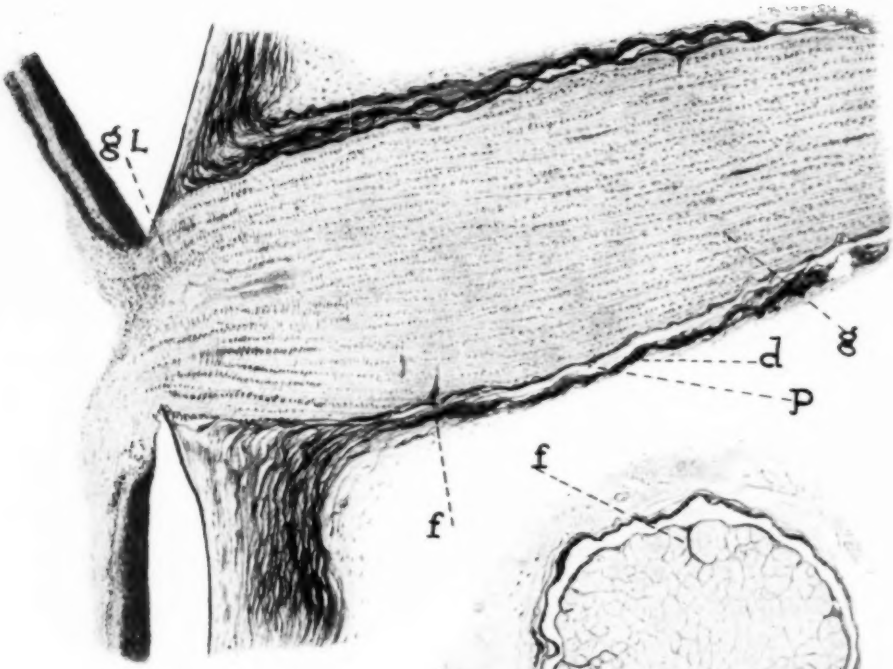

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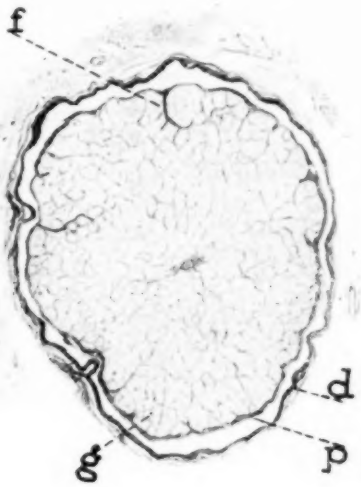

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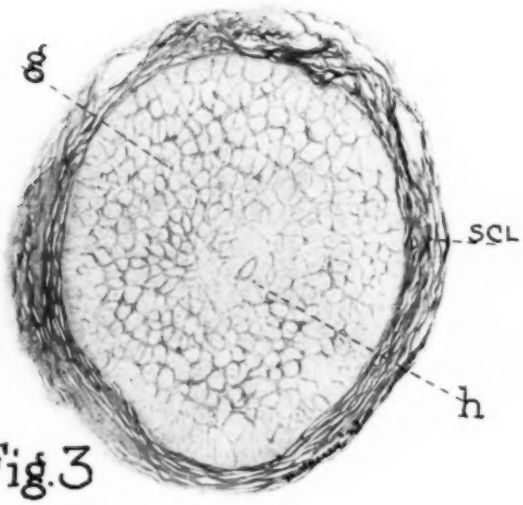

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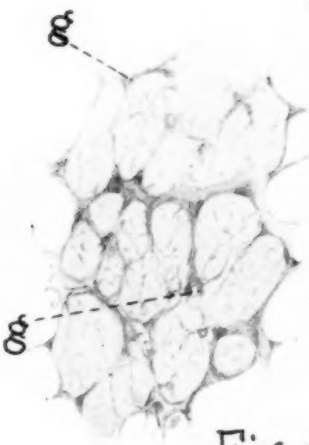

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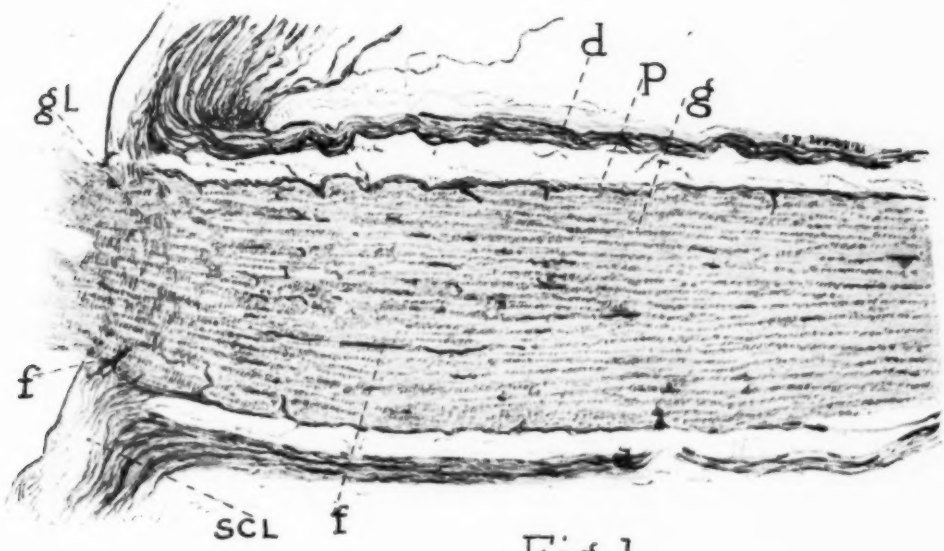

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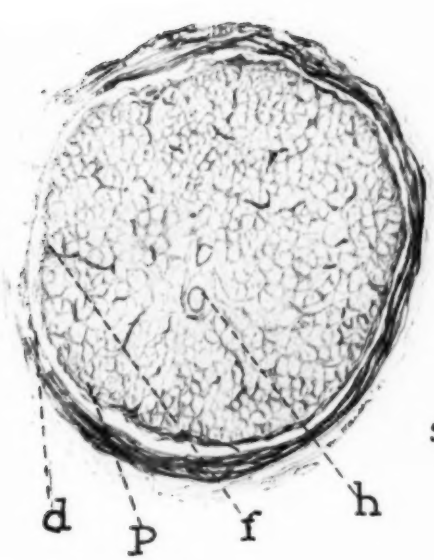

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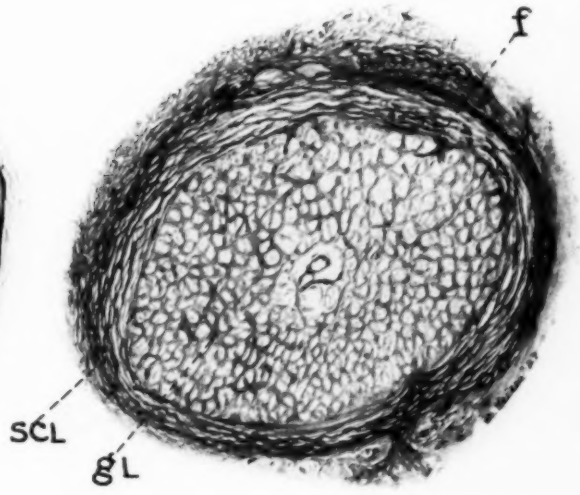

Fig. 3.

DEVELOPMENT OF OPTIC NERVE AND LAMINA CRIBROSA

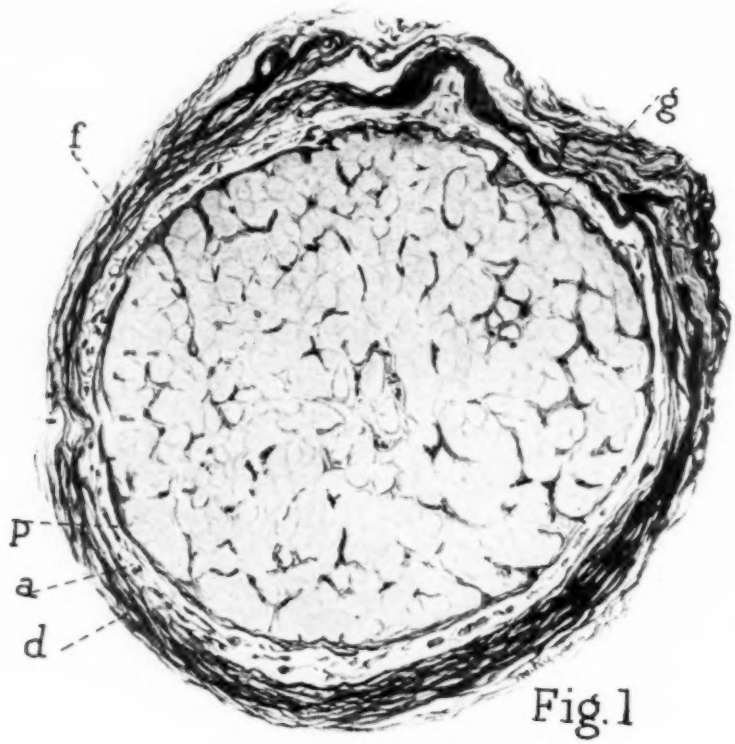

Fig. 1

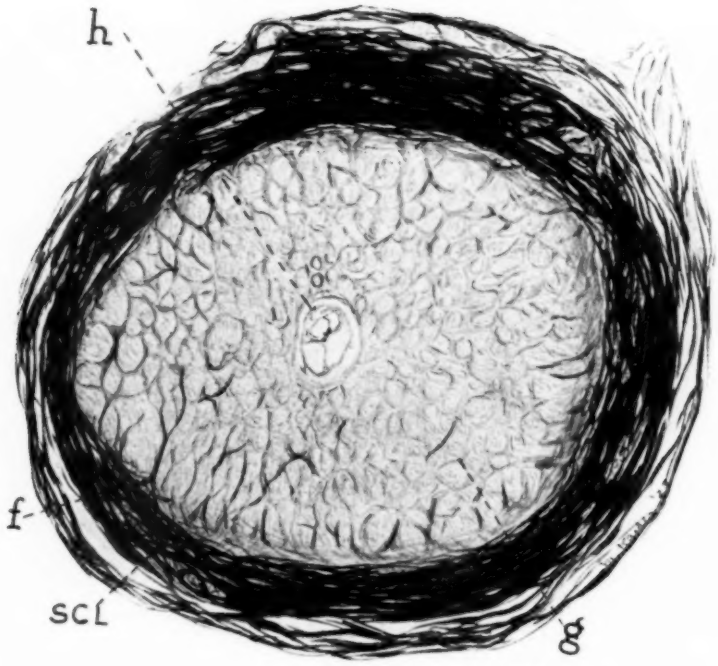

Fig. 2

DEVELOPMENT OF OPTIC NERVE AND LAMINA CRIBROSA

LATE 4

AMERICAN JOURNAL OF OPHTHALMOLOGY

Vol. 8 JANUARY, 1925 No. 1

THE DEVELOPMENT OF THE CONNECTIVE TISSUE FRAMEWORK OF THE HUMAN OPTIC NERVE WITH ESPECIAL REFERENCE TO THE LAMINA CRIBROSA.

HENRY C. HADEN, M. D.

HOUSTON, TEXAS.

This paper is based on the histologic study of nine human fetuses, from 45 mm. to 500 mm. in length. The sections were stained to differentiate glial from fibrous connective tissue. They show that the optic nerve is originally wholly ectodermal. Later it becomes invaded by fibrous connective tissue elements from the mesoderm of the anlage of the pial sheath. The fibrous connective tissue of the lamina cribrosa is not a growth forward of the septa of the nerve, but a direct ingrowth of the collagenous fibers of the sclera.

Until comparatively recently the lamina cribrosa was thought to be produced by the fibers of the optic nerve passing thru the anlagen of the choroid and sclera as they grow into the optic stalk. In a standard text book of embryology of so recent edition as 1921, the following statement is made:

"As the mesodermal anlagen of the choroid and sclera are present before the nerve fibers begin to grow into the optic stalk, the fibers must pass thru these two coats on their exit from the eye. There results the fenestrated crossing of the optic nerve by these two coats, known as the lamina cribrosa." (Bailey and Miller)

Like statements are found in other quite recent publications of authoritative writers. That this is not only incorrect, but that it would be a mechanical impossibility, may be readily understood if one will but recall that the optic stalk is but a part of the optic vesicle and that there is no separation in their continuity after the invagination, or at any time, and that the fibers grow back from the cup *thru the substance of the wall of the stalk*. Therefore, as the anlagen of the choroid and sclera are exterior to the optic cup and stalk, it would be impossible for the nerve fibers to grow thru them in such manner.

As the lamina cribrosa occupies a unique position in the eye which causes it to participate in many pathologic processes of grave consequences, a knowledge of its nature should be of value. With this thought in mind, some few years ago I undertook an investigation into its development; but soon found that to do this meant a knowledge of the history of the development of the whole fibrous framework of the optic nerve.

I, therefore, present here its complete history. The material used is my own, and all the sections and paintings are original to this article. My study was made from nine human fetuses, ranging in length from 45 mm. to 500 mm. I have endeavored to show the various phases of its development and trace the changes step by step that lead up to the last stage in the growth of the sustentacular tissue of the optic nerve, *the appearance of the fibrous connective tissue fibers in the lamina cribrosa.* I have sought to do this in a simple, straightforward way, in order that those who have neither the time nor the inclination to do this work themselves, nor the patience to wander thru the mazes of controversy in foreign literature, may have the privilege of seeing this very interesting picture at a glance.

It will be my endeavor to demonstrate the following:

1st. That the lamina cribrosa is primarily glial, of ectodermal origin.

2nd. That toward the end of (or in the later months of) fetal life the posterior layers (brainward) of the lamina show the presence of connective (mesodermal) tissue.

3rd. That this connective (meso-

dermal) tissue is not a growth forward of the fibrous septa of the optic nerve, but is a direct inward growth of the collagenous fibers of the anterior layers of the sclera.

4th. That the anterior layers of the lamina (choroidal portion) are *purely glial* (ectodermal) and *remain so in adult life.*

In order to understand what is to follow, it is necessary to have some knowledge of the development of the ectodermal, or primary, framework of the optic nerve. As this paper and these specimens begin shortly before the appearance of the mesodermal tissue (that is, at about two and one fourth months, 45 mm., g. l.), I shall quote from a resumé of the work of Dr. Arthur Robinson on the development of the optic nerve in mammals, which presents a clear explanation of the origin and development of the glial framework of the optic nerve:

"As the fibers grow from the nerve cells with which they are connected, they pass into the substance of the stalk, forcing their way between its constituent parts, and ultimately acquiring an intercellular position. It is not however possible to state definitely that they possess such a position on the first entry into the stalk, for there are indications that in the early stages the cell territories of the stalk are not defined; or, in other words, that the stalk is a hollow nucleated plasmodium, and that the separation of the protoplasmic mass into cell areas is possibly initiated by the entrance of the optic nerve fibers into it. In support of this possibility, it is to be remembered that it is only as the nerve fibers increase in number that the cell territories become defined, and that the constitutent cells are never completely isolated from each other, for they remain connected by anastomosing processes which limit the areas thru which the nerve fibers pass; but however this may be, there can be no doubt about the final result, which is that the nucleated protoplasmic walls of the optic stalk become directly converted into the sustentacular tissue of the optic nerve exactly in the same manner that the spongioblasts of the spinal cord and brain are converted into the neurospongial substance; *and in the first instance at all events the mesodermic tissue takes no part in the formation of the optic nerve, tho at a later period a mesodermal invasion of the nerve must occur in association with the ingrowth of the vessels into the nerve substance.*"*

I shall now endeavor to show how this mesodermal invasion of the nerve takes place by describing the appearance of the optic nerve in each of the nine fetuses that I have studied. *Specimen 1, Plate 1, Figure 1, Stain H and E, Objective 16 mm., Ocular 10, A Human Fetus 45 mm., greatest length; approximate age two and one half months.*

The optic nerve was composed of a mass of fibers running among numerous glial cells. The glial cells were beginning to show some tendency to regularity of arrangement, but were not in sharply defined rows. At this age there was no mesodermal tissue in the optic nerve other than that which accompanied the hyaloid artery. There were no blood vessels in it other than this one, except an occasional ill-defined capillary in the brain end. The nerve was immediately surrounded by the anlage of the pial sheath, which was composed of minute capillaries and certain cells with large, pale granular nuclei, which were fibroblasts.

A careful study made of the border of the nerve showed that its most intimate covering, or membrane, was composed of glial cells placed side by side. This was the anlage of the peripheral glial mantle. And when this layer was followed forward to the globe, it was seen that the cells of this layer were continuous with the pigmented layer of the retina. This layer was best seen in that part of the nerve near to the retina, in the region of what will later be the choroidal canal. In the sections toward the brain it was more difficult to see, because it had—at even this early age—become cov-

*On the Formation and Structure of the Optic Nerve, and its Relation to the Optic Stalk, By Arthur Robinson, M. D., The Owens College, Manchester, England. (Journal of Anatomy and Psysiology, Vol. 30.)

ered by connective tissue cells and capillaries of the pial sheath.

At this age the sustentacular tissue, framework or septa of the optic nerve was purely glial, and had been developed from the plasmodium of the optic stalk. It was an imperfectly developed and irregularly arranged syncytium of glial cells. In older fetuses these cells will become perfectly and beautifully arranged, as will be seen in the cross section of the 97 mm. fetus.

Specimen II, Plate I, Figures 2 and 3. Stain H. and E. Obj. 16 mm. Oc. 10, Obj. 2 mm. Oil Immersion Oc. 10. A Human Fetus, 67 mm. g. l. Approximate age, two and three quarter months.

There was a marked change in the appearance of this fetus from that of the 45 mm. The glial nuclei showed a greater tendency to be arranged in parallel longitudinal rows, which marked the divisions of the funiculi; but they were still not quite regular, except some of those in the distal (retinal) end, which were seen to lie with their long axes at right angles to the nerve fibers and were in definite parallel lines. This constituted the anlage of the lamina cribrosa. (See Plate I, Figure 2 at gl.)

The most striking change in this nerve from the younger one was the appearance of capillaries in its substance (c). These were most numerous in the proximal (brain) end of the nerve. Their walls were delicate single layers of endothelial cells. They were so delicate that at times one saw blood corpuscles without being able to distinguish the vessel wall. Many of the capillaries, or endothelial tubes, could be seen entering the nerve from the pial sheath, and sections of others were seen scattered very generally thruout the nerve (Plate I, Figure 2 at c). It was seen that none of these capillaries originated within the optic nerve, but arose from the surrounding mesoderm (anlage of pial sheath) and grew into the nerve. Their advent represents the first invasion of the mesoderm into the substance of the optic nerve. They contained blood corpuscles and showed endothelial cells in advance of them—endothelial budding. They were surrounded by many cells with large pale nuclei, which, if compared with cells of the neighboring loose connective tissue, would be seen to be fibroblasts. (Plate I, Figure 3 at c and f b. Obj. 2 mm. Oil Immersion Oc. 10.)

Within the optic nerve the processes of these cells might only occasionally be seen, as they were hidden by the nerve fibers and glial cells. I shall show in succeeding pictures that these capillaries, accompanied by the fibroblasts grow and climb around the glial cells which form the partitions between the bundles of nerve fibers; and gradually, as the young connective tissue fibrils develop from these capillaries and fibroblasts, they enmesh the glial trabeculae and finally replace them and become the connective tissue septa.

The dural sheath of this fetus was composed of delicate wavy collagenous fibrils which stained differentially. The fibers of the pial sheath stained with the Van Giesen stain the same as the dural, only more intensely red. The mesodermal tissue accompanying the hyaloid artery showed distinct strands which stained bright red. Other than that accompanying the hyaloid artery, *there was no mesodermal tissue within the nerve of this fetus, sufficiently developed to stain differentially.*

Specimen III, Plate II, Figure 1. Stain The Van Giesen. Obj. 16 mm. Oc. 10. A Human Fetus, 97 mm. g. l. Approximate age three months. Long. Sec.

In this fetus was seen the next step in the development of the framework of the nerve, which was the appearance of collagenous fibrils among the cells of the glial septa. Longtitudinal sections stained by the Van Giesen method showed greater development of the dural and pial sheaths than was seen in the two preceding fetuses. A number of collagenous fibrils were seen running into the nerve from the pial sheath, and others were seen lying along the divisions between the funiculi. The glial cells were now lined up in parallel, longitudinal rows, which represent the septa between the bundles of nerve fibers. The hyaloid artery and vein were surrounded by connective tissue fibrils. Capillaries and blood corpuscles were seen in among the glial cells in nearly every one of the septa. The glial nuclei, which were in the anterior or retinal end of the nerve opposite the

anlage of the sclera, lay with their long axes at right angles to the nerve, and were arranged in numbers of parallel rows; these form the anlage of the lamina cribrosa and they were very distinct. (Plate II, Figure 1 at gl.)

Plate II, Figure 2, Stain The Van Giesen, Obj. 16 mm. Oc. 10. Same Fetus, Cross Section of Orbital Portion of Optic Nerve.

In cross sections of the fellow eye of the fetus it was seen that the sustentacular tissue, or frame work, of the nerve consisted of glial cells and their processes, within the spaces of which were seen other glial cells and fine glial fibers and nerve fibers. Capillaries in cross sections lay at the cellular junction of the glial septa in association with the nuclei of the glial cells. Longitudinal sections of capillaries leading into the nerve from the pial sheath were seen, and occasionally a red streak of connective tissue fibrils appeared running from the pial sheath into the nerve along the septa (Plate II, Figure 2, at f.) Small pieces of red fibrils which showed no connection with the sheath were scattered about in among the septa, and delicate webs of connective tissue fibrils were seen about the glial septa. The hyaloid artery and vein were surrounded by delicate connective tissue fibrils.

The serial sections of this nerve, studied from the proximal or brain end, near the optic foramen and reading forward toward the eye, presented the general appearances just described. Only occasionally could the peripheral border of glial cells (peripheral glial mantle), which separate the nerve fibers from the pial sheath, be seen, as it was so intimately associated with the sheath that it was obscured by its connective tissue fibrils and capillaries. As the entrance of the nerve into the eye was approached, the appearance of the glial septa changed very much. They became thicker and coarser. This change became very marked after the nerve entered the scleral canal—that is, after the pial sheath and intervaginal space were no longer seen. (Plate II, Figure 3 at g.)

Plate II, Figure 3, Stain The Van Giesen, Obj. 16 mm., Oc. 10. Same Fetus, Cross Section thru Lamina Cribrosa.

This is the position that corresponds to the parallel rows of glial nuclei with their long axes at right angles to the nerve seen in the longtitudinal sections —in other words, the position of the lamina cribrosa (Plate II, Figure 1 at gl.). Here the glial trabeculae were very coarse and thick and the capillaries were endothelial tubes with no connective (mesodermal) tissue fibrils. With higher magnification, the nuclei, cell bodies and processes of the glial cells, showed very well.

Plate II, Figure 4, Stain The Van Giesen, Obj. 2 mm. Oil Immersion, Oc. 10. Detail of Glial Cells in Same Section as Last. Higher Magnification.

The cells had large densely staining nuclei and ray like processes which anastomosed with those of other cells. These glial cells and their processes formed the primitive lamina cribrosa. In the succeeding pictures it will be shown that the posterior layers of these anastomosing cells will be invaded by mesodermal (connective) tissue from the sclera. But the anterior layers remain purely glial thruout fetal life, and present the same characteristic arrangement. The peripheral border of glial cells which surrounds the nerve and sends processes into it was well seen. As heretofore stated, the peripheral border of glial cells is continuous thruout the whole surface of the nerve in all probability, but cannot always be demonstrated because of its intimate association with the pial sheath.

Note that the connective tissue of the sclera stained very red. It could be very positively seen that there was no tissue that stained red in the position occupied by the lamina. As the nerve fibers had grown into the nerve long before this age, it can be seen beyond question of doubt that they did not pass thru the anlagen of the choroid and sclera to make the fenestrated appearance known as the lamina cribrosa.

Specimen IV, Stain The Van Giesen. A Human Fetus, 105 mm., g. l. Approximate age three and one half months.

This fetus differed very little in appearance from the 97 mm. fetus. There were more connective tissue fibrils which stained red accompanying the little blood vessels, and the glial cells showed more

perfectly. The lamina cribrosa was composed of glial cells and their processes and there was no mesodermal tissue in it.

Specimen V, Plate III, Figure 1, Stain The Van Giesen, Obj. 16 mm., Oc. 10. A Human Fetus, 180 mm., g. l. Approximate age four and one half months.

In this fetus the scleral and pial sheaths showed greater development. Within the nerve bright red lines of connective tissue fibrils were seen in many places between the bundles of nerve fibers. Where they lay the glial nuclei were not seen, but at the end of the red streaks they reappeared and continued to be seen as the septa. The glial lamina cribrosa was well developed. The cell processes were drawn out thickly, and lay in numerous parallel rows at right angles to the nerve opposite the level of the sclera. It was seen that the immediate periphery of the nerve at this place was bordered by a row of glial cells, whose processes might be seen to run inward to assist in forming the lamina. When this row of cells was traced forward, it was seen to curve over the edge of the lamina vitrea choroidea and become continuous with the retina, as in the younger fetuses. One bright red bundle of connective tissue fibrils was seen running from the sclera into the posterior layer of the lamina. This was the first evidence of mesodermal tissue in the lamina and it is apparent that it comes from the sclera.

Plate III, Figure 2, Stain The Van Giesen, Obj. 16 mm., Oc. 10. Same Fetus, Cross Section of Orbital Portion of Fellow Eye.

Cross sections of the orbital portion of the nerve of the fellow eye of the same fetus, stained with the Van Giesen stain, showed numbers of bright red bands of connective (mesodermal) tissue forming septa, outlined by capillaries. As the sections approached the globe the connective (mesodermal) tissue within the nerve became less. Cross sections thru the nerve at the level of the sclera and in advance of the intervaginal space (Plate III, Figure 3, same stain and magnification), showed strong trabeculae formed by glial cells and their processes, the lamina cribrosa (stained brown). The nerve was here surrounded by a dense border composed of glial cells, immediately surrounding which were the circular, red fibers of the sclera. The lamina cribrosa was as yet free from connective tissue, except for one or two strands which ran from the sclera into its posterior layers (Plate III, Figure 3 at f). Similar strands were seen in the longitudinal sections of the fellow eye of the same fetus (Plate III, Figure 1).

Specimen VI, Stain The Van Giesen. A Human Fetus, 190 mm., g. l.

The appearances of sections of this fetus were practically the same as those of the 180 mm. fetus just described.

Specimen VII, Plate IV, Figure 1. Cross Section of Orbital Portion of Nerve. Stain The Van Giesen, Obj. 16 mm., Oc. 10. A Human Fetus, 360 mm. g. l. Approximate age seven months.

In this specimen the sheaths of the nerves were much more developed and the vessels running from the pial sheath into the nerve were larger, and had more fibrous tissue in their walls than in the younger specimens. As was seen in the younger fetuses, these vessels passed between each funiculus, or bundle of nerve fibers. Associated with them were many bundles of connective tissue fibrils, and the glial septa between the funiculi in the greater portion of the nerve had been enveloped and partially replaced by the young connective tissue (Plate IV, Figure 1 at f and g). The fibrils of this connective tissue were curly, loosely meshed, and took on a characteristic purplish red color when stained by the Van Giesen method. Within the funiculi were numerous glial cells and fibers among the nerve fibers. Seen in longitudinal section, the fibrous septa were not continuous and the gaps within them were filled in by glial tissue, the same as is seen in postnatal life.

Plate IV, Figure 2, Cross Section of Same Nerve thru the Posterior Layers of the Lamina Cribrosa, Stain The Van Giesen, Obj. 16 mm., Oc. 10.

As the cross sections of the optic nerve of this fetus entered the posterior pole of the globe, and they were seen to have passed beyond the anterior end of the intervaginal space; or in other words, were in advance of

the anterior end of the pial sheath, a great change was seen to have occurred in their appearance. The septa had lost the purplish stained, wavy, fibrous connective tissue, and were now composed of coarse bands of glial tissue. These were interlaced by straight fibers of bright red stained, fibrous connective tissue; which were seen to be directly continuous with the fibers of the anterior layers of the sclera (Plate IV, Figure 2 at f and g). The sclera had identically the same intense red appearance as these fibers. The fenestrated appearance just described was the posterior portion of the lamina cribrosa. It could be very positively seen that the connective tissue fibers which were forming part of its structure were a direct ingrowth of the fibers of the sclera, and not a forward growth of the connective tissue septa of the orbital portion of the nerve. The structure and staining qualities of the fibrous tissue in the two positions were seen to be quite unlike. Where the connective tissue fibers of the lamina crossed one another, their intersections were seen to be angular and not curved. Immediately surrounding the position of the lamina, the scleral fibers were seen to be arranged in circular formation. The longitudinal sections of this nerve showed that the lamina was bowed, or curved, with the convexity toward the brain, as seen in postnatal life. As the cross sections progressed forward, and the level of the choroid was reached, all the red staining connective tissue fibers had disappeared completely. The lamina now was composed entirely of glial cells and their processes, and glial fibers. It presented almost an identical appearance as that of the lamina of the 97 mm. fetus, only the septa were coarser. This is the choroidal portion of the lamina and it is never invaded by fibrous connective tissue, but remains purely glial thruout postnatal life. It may be considered a later stage of the original network of glial cells with their anastomosing processes, which arrangement was inaugurated by the invasion of the optic nerve fibers into the optic stalk. In the cross sections of this specimen at the level of the lamina, the peripheral border of glial cells and the inner border of glial cells (those surrounding the central vessels) showed very distinctly.

Specimen VIII. A Human Fetus, 400 mm., g l. Approximate age eight months.

The sections of this fetus had practically the same appearance as those of the 360 mm. fetus, except that the fibrous connective tissue, both of the orbital portion of the nerve and the lamina cribrosa, was greater in quantity and coarser in texture.

Specimen IX. A Human Fetus, 500 mm., g. l. Approximate age nine months.

The fetus was presumably just at term, but had had no postnatal life. Sections of the optic nerve stained by Weigert's method demonstrated that there were no medullary sheaths to the nerve fibers. The specimen was wonderfully preserved. The fibrous connective tissue of the septa of the orbital portion of the nerve was in more compact bundles than in the 400 mm. fetus, and that of the lamina not so thin and delicate. Sections stained with Mallory's glial stain showed clearly the intermingling of the glial fibers and the connective tissue fibers of the posterior layers of the lamina, but there were no fibrous connective tissue fibers among the glial fibers of the choroidal portion of the lamina.

RESUMÉ.

The primary framework or sustentacular tissue of the optic nerve is derived from the ectoderm; and is composed of glial cells and their processes. By the continuity of the processes with one another an arrangement similar to a bundle of tubes is made, within which lie the optic nerve fibers. The ectodermal (glial) framework may be likened to a pattern or skeleton upon which a permanent structure is later to be built. Prior to the middle of the third month there is no mesodermal tissue within the optic nerve, other than that incarcerated with the hyaloid artery. At this age the mesoderm begins its invasion. The first signs are a few capillaries which arise within the anlage of the pial sheath and pass thru the peripheral

layer or covering of glial cells of the optic nerve and enter it. They are endothelial tubes which are accompanied by fibroblasts. The capillaries, after entering the nerve, follow the direction of the ectodermal partitions composed of glial cells, and their processes, and gradually increase in number until a capillary is found at nearly every glial partition. As this is taking place, the accompanying fibroblasts develop connective tissue fibrils, which form delicate webs of tissue about the capillaries. As this fibrous connective tissue increases, it envelops the glial cells and processes which form the glial septa, and by the seventh month nearly all the septa between the funiculi are fibrous connective tissue. This metamorphosis apparently does not occur in unbroken lines, for even in adult life there are gaps in the connective tissue septa which are filled in by glial cells.

As early as two and three quarters months the glial nuclei in the distal or retinal end of the nerve, opposite the location of the anlage of the sclera, assume an appearance and position different from those in the rest of the nerve. Their processes are coarser and form in this place a strong network which is the anlage of the lamina cribrosa. This portion of the glial sustentacular tissue is not invaded by fibrous connective tissue until at a much later period than that of the older parts of the nerve. When ultimately it does appear here it is a direct ingrowth of fibers of the sclera, and it occupies only the posterior (brainward) layers of the lamina. The anterior or choroidal portion of the lamina is never invaded by connective tissue, but is composed of glial cells and their processes, which are arranged just as they were in early fetal life, and should be considered as a persistence of the syncytium of ectodermal cells which formed the original framework of the optic nerve. At the end of fetal life the fibrous connective tissue fibrils of the lamina are thin and delicate, but very soon after birth they become coarser and apparently of great strength in order to withstand the strain entailed by the movements of the globe.

AN OPINION AS TO FUNCTION.

The fibrous connective tissue portion of the lamina serves to close partially the gap in the sclera made by the scleral canal. It also prevents changes in the shape of the posterior pole of the globe when it is moved by the external ocular muscles. The scleral fibers of the lamina, passing into the nerve between its funiculi, serve to hold it in fixed position (anchored to the sclera, in other words) during the movements of the globe. It does not begin to develop until shortly before birth, because there is no need for it until then, and at birth the collagenous fibers are comparatively thin and delicate. After birth they increase in thickness and strength to keep pace with the increased movements of the eyes, and continue to grow until in adult life. In adult life they have become thick and strong, in order better to resist the strain of the pull upon the sclera during convergence and the ocular movements. If it were not for the anchoring of the nerve by the connective tissue lamina, there would be a disturbance in vision caused by the pulling of the nerve upon the retina when the eye changed positions.

My thanks are due to Mr. R. J. Davies,, Technician of the biologic laboratories of Rice Institute, for the excellent manner in which he prepared the sections for this study, and for his many valuable suggestions.

THE OUTSTANDING BEAM OF THE AQUEOUS FLUID.

BASIL GRAVES, M. C.

LONDON, ENGLAND.

This paper describes the exact technic by which the path of a minute brilliant beam is rendered visible in the normal aqueous. Familiarity with this normal appearance is the basis for judging pathologic changes observed in microscopy of the living eye. Cases illustrating different changes of the kind are mentioned. The normal visibility of the aqueous under proper technic has already been referred to in a paper about to be published in the British Medical Journal.

The normal body fluids, e. g. the cerebrospinal fluid, are not optically empty. When a sharply defined intense beam of light passes thru such fluids the track of the beam is visible. Its visibility is due to (1) scattering of light—by minute particles whose

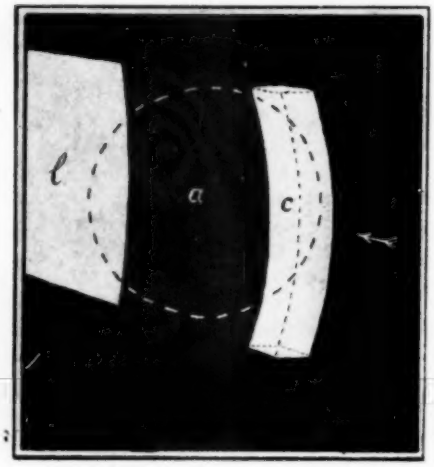

Fig. 1.—Slit beam in clear media; c, cornea; a, aqueous; and l, lens. The circle indicates part included in the field of vision at one time.

size and distance of separation is below the lowest limit admitting of resolution—which gives rise to a homogeneous ghost like shaft, spoken of as the "outstanding beam:" and (2) diffraction by particles of such a size that they are individually visible as such—cellular or amorphous small floating bodies. It is not the purpose of this article to deal with these, but only with the outstanding beam of the aqueous fluid whose variation, under pathologic conditions, is a very interesting feature in microscopy of the living eye. This outstanding beam can be seen in the normal aqueous fluid by suitable methods to be described; and its visibility is increased in various pathologic conditions, an account of which has not, as far as I am aware, yet been forthcoming. The recognition of this is not a scientific curiosity; it is, I believe, of more practical clinical significance than the de-

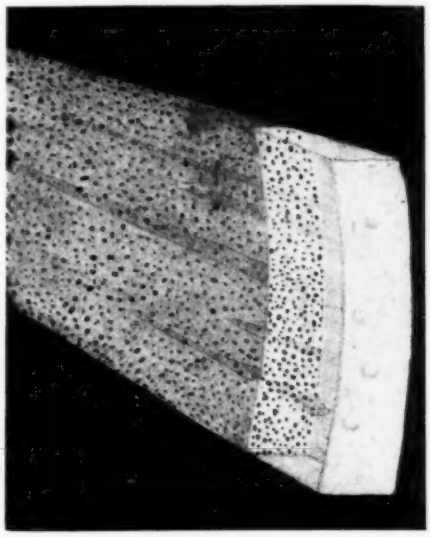

Fig. 2.—Slit beam passing thru pathologic aqueous showing many straw colored granules such as were also deposited on the back of the cornea. Also shadow streaks due to irregularities of the corneal surface.

tection of visible floating particles in the aqueous fluid, because it occurs with much greater frequency than the pathologic presence of these. To know when this outstanding beam is pathologic it is very necessary to be able to recognize it and to be familiar with its appearance in the normal eye.

The visibility of the beam in the normal aqueous fluid is very faint and only when the aqueous fluid is very abnormal, so that from gross alteration of the colloid, physical and chemical constituents it becomes relatively highly relucent, can the passage of the beam be seen by the method indicated in Fig. 1, where the slit beam path covers, in width, more than the diameter of the visual field (*c* representing

cornea, *a* aqueous fluid, *l* lens, and the dotted circle the limit of the field of observation). A more delicate method is so to arrange the slit beam that its upper or lower edge (Fig. 3) crosses the visual field, and thereby to secure the necessary contrast at the junction of illuminated and unilluminated fluid. This method, tho an improvement, is nevertheless not a delicate one for observing the relucency of the aqueous fluid; for one reason, too much light is projected into the observed eye and is diffusely reflected in and about the anterior chamber, with the result that visual contrast is impaired. The method is, however, applicable to appreciating increase of visibility of an outstanding beam which is pathologic to a fairly definite degree. Such pathologic pictures as that of Fig. 2 are already forthcoming commonly in relation to uveitis in its severe—for example sympathising—forms. Now it so happens that there are many clinical conditions in which the visibility of the aqueous beam is pathologically increased only to a slight degree; and the observer will fail to appreciate this unless he has well accustomed himself to see the beam, just visible, as it traverses the normal aqueous fluid. To see this, the following technic, which I have employed for a long time, is, I think, the only one which is satisfactory; the vertical diameter of the slit beam is cut down and a beam of small cylindrical diameter is used.

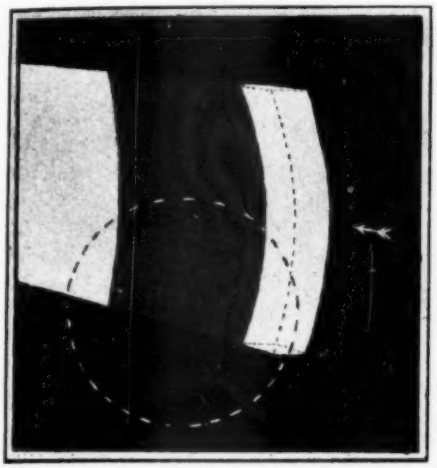

Fig. 3.—Slit beam crossing upper part of field of vision indicated by the circle.

With the ordinary nitra slit lamp, using the 10 mm. dot aperture, the observer may see, as shown diagrammatically in Fig. 4, the path of the beam traversing any normal aqueous fluid, if he attends to the following points: It is essential that the filament of the nitra lamp should be loaded to the maximum of its current capacity, which is more than 8 volts; this generally necessitates altering the set-stop, inside the adjustable resistance, from the position it is in when sent out by the makers, and placing it where it allows the filament, as judged by naked eye inspection, to glow with very vivid intensity. These lamps will carry a very heavy load, and they are not expensive. Only a few seconds are necessary for the observer's dark adaptation, but the room must be really dark. The patient should look slightly up at the observer's forehead. The illuminating beam, on its way to the eye, must not pass thru lashes; or the lashes, when long, may be accidentally illuminated in another way, by the beam which is reflected from the corneal epithelium glancing sharply upwards thru them: in either case a reflected image of the illuminated lashes may be seen as if in the anterior chamber, confusing the view. The observer should choose an angle of il-

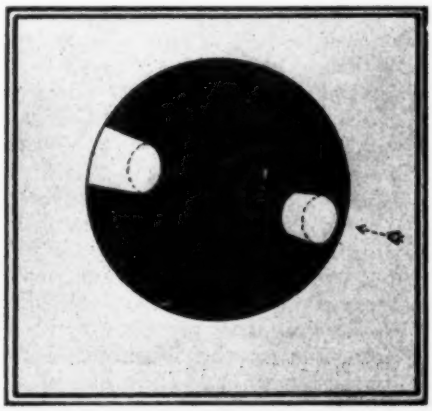

Fig. 4.—Path of small beam of concentrated light visible in normal aqueous. The white portion of the beam represents the cornea on one side and part in the crystalline lens on the other. The narrow faint grey portion gives the appearance of the beam in the normal vitreous.

lumination which will not allow the beam reflected from the corneal epithelium to strike the microscope objective lenses. The beam should be accurately focused in the middle depth of the aqueous fluid—that is, midway between the cornea and the anterior lens capsule. After traversing the aqueous fluid, this beam should not strike the iris—a not uncommon error which gives rise to much diffuse reflection within the anterior chamber—but should pass on thru the pupil into the lens, for which reason it is easier to observe the relucency of the normal aqueous fluid when the pupil of the observed eye is dilated. The observer, using preferably oculars No. 2 and objectives a2, should see the small round corneal beam just on the one side of his chosen field and the beam as it enters the lens on the other side, the illumination being focused midway between these two levels; he will then bring the microscope into focus on the illuminated aqueous fluid by first focusing on the illuminated corneal patch or on the illuminated anterior lens capsule, then deliberately rocking the microscope a little forwards or backwards as the case may be, to an extent which becomes automatically accurate with practice. The necessary adjustments thus being made, the observer need attend to only one or two points in order to see the beam traversing the normal aqueous fluid: he should move the slit lamp arm slowly up and down so as to give slight movement to this faint beam which thereby will be seen the more easily; and, at least when a beginner, he should take observations with his own more appreciative perimacular field, by directing his gaze deliberately a little above or below the level at which he seeks to see the beam.

The normal aqueous beam is much more readily visible when the arc slit lamp is used, in which case it is better to work with the 0.5 mm. dot aperture. I am not sure that the arc lamp is an advantage for thus judging pathologic increase, because the mere fact of the nitra lamp beam being only just visible in the normal aqueous fluid makes it all the easier to form an opinion on whether or not its visibility is pathologic. If the observer works with the arc lamp for this purpose he should thoroly acquaint himself with the degree of visibility of the beam in the normal aqueous fluid.

When the arc slit lamp is thus used, occasional isolated illuminated particles may be seen to cross the beam track even in an eye which may be presumed to be normal; I believe that such particles, when very few and when unaccompanied by any increase in the normal visibility of the outstanding beam, are not necessarily indicative of past or present intraocular trouble. I have seen such a particle perform a gyratory motion about the path of the cylindrical beam: possibly this may afford evidence of a local convection effect due to the warmth of the rays.

It is not the purpose of this article to discuss the variety and significance of visible discrete particles within the aqueous fluid, but some mention of them is desirable. With certain reservations, these are generally seen better when illuminated by the ribbon like slit beam than by the cylindrical dot beam. The slit should be opened wide, and in certain instances it is actually better to let the beam be directed upon the iris near the pupil margin; but this is not always the case—it is connected with the diffuse back reflection of light. Granules under direct illumination* are more readily visible by means of the arc lamp than the nitra lamp. A certain method of retroillumination*, which depends on axial observation along the efferent beam reflected from the posterior lens capsule, is described in the Brit. Journ. of Ophthalmology for November, 1924; if this method is used for observation of the aqueous fluid the presence of granules is often revealed, with the nitra lamp, on occasions when they are not readily visible by direct illumination. The detecting, or not, of granules in the aqueous fluid depends on the methods of examination.

These particles may be blood cells and pigment cells; but they are also often amorphous material. It is not

*These and other terms are defined in the Transactions Ophthal. Soc., vol. XLIII, 1923, "Microscopy of the Living Eye."

justifiable in ordinary clinical slit lamp work to try to discriminate histologic types of cells in the anterior chamber. When particles which are very small indeed are thus directly illuminated, tho it may be possible to say that they are colored and are not white, the variety of color commonly cannot be identified. When such particles are seen after, e. g., a blow on the eye, if they are very sparse and fine it may be merely random guessing to say that

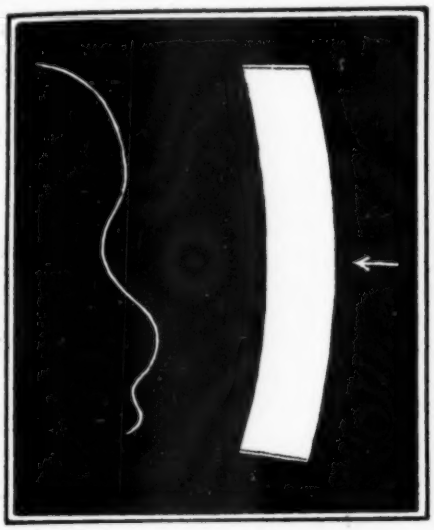

Fig. 5.—"Impact line" on face of prolapse of the vitreous into the aqueous. This is the curved line to the left, the broad arc to the right representing the illuminated section of the cornea.

they are yellow-red in color and that they are therefore red blood corpuscles. On this account, when speaking of particles seen in the anterior chamber of recently injured eyes, I prefer to use the term granular débris (uveal pigment may be present as well as blood material) than to speak of seeing blood corpuscles. As will be seen presently, under the subject of concussion injury to the eye, particles, very evident as they circulate in the anterior chamber, may contribute to the greatly increased visibility of the beam; but, long after their disappearance, the outstanding beam may for months remain pathologically visible —due, it may be presumed, to the presence of colloidal material derived from the blood.

Other visible particulate bodies in the anterior chamber, besides those derived from blood and from uveal pigment, may come from the lens—e.g. disseminated matter after rupture or needling of the lens. Vitreous within the anterior chamber may increase the visibility of the beam: a fluffy "dilute" vitreous material, perhaps mixed with lens debris, may cause increase in the visibility of the posterior part of the aqueous beam, after cataract extraction and needling. On the other hand a prolapse of compact unpigmented vitreous into the anterior chamber may readily pass undetected—especially when the slit beam is used—unless the beam is made exceedingly narrow and is critically focused so as to form an "impact line" on the face of the prolapse, as, in Fig. 5 (for a description of impact lines, occurring at the face of demarcation between two media of different refractive index, see "Slit lamp features of a small tumor at the limbus," Trans. Ophthal. Society, Vol. XLIV, 1924). Such a vitreous line, accidently seen, can be mistaken by a beginner to be a variety of reflex image in the anterior chamber. When a wide slit beam is used I have known an illuminated tag of persistent pupillary membrane, torn and waving free in the anterior chamber after an injury, to be mistaken for prolapsed vitreous.

The technic described above for observation of the normal aqueous beam is followed by making clinical observation on pathologic cases of the type now to be described. Occasionally there are difficulties: If from infiltration of the upper and central parts of the cornea, as in some cases of interstitial keratitis, the anterior chamber can be viewed only thru the lower periphery of the cornea, it may be difficult to judge of pathologic change of the aqueous beam because the beam must needs impinge on the iris. On the other hand, in eyes in which the lens is opaque and very relucent, it may be better to direct the beam on to the less relucent iris; but such cases are unsatisfactory for appreciating fine changes. Other unsatisfactory cases are those with a shallow anterior chamber and small undilatable pupil,

e.g. eyes with iris bombé, where an effort must be made to keep the follow thru track of the beam within the limits of the small pupil.

In eyes which have been injured, the factors on which increased visibility of the aqueous beam depend are hemorrhagic or serous effusion from the vessels; the presence or absence of disseminated lens matter; and, later, the occurrence or not of inflammatory trouble, e.g. iritis. The beam may be especially evident after injuries requiring magnet operation for removal of a metal foreign body, particularly if the lens has been injured or if there has been hemorrhage. I found the flare very visible in a man ten weeks after removal, by magnet, of a foreign body which had made a hole in the iris and had caused minimal damage to the anterior lens capsule—resulting in a small local opacity—tho the eye showed no evidence of ensuing iritis. But periods differ: I have seen the aqueous beam normal in a man who eight weeks before received a wood chopping injury which perforated the cornea and iris. The beam is generally found to be very evident in the type of injured, perforated eye which comes to enucleation—e.g. one with vascularized iris adherent to a lens whose capsule has been badly torn. Eyes which have a clean accidental penetration of the cornea only (e.g. by a piece of wire) examined a day or so after may be found to show slightly increased visibility of the aqueous beam. Such eyes, however, examined some weeks afterwards, having run an uneventful course, may often be found to show a normal aqueous beam.

In a case in which, on the center of the cornea, there was a small superficial septic spot which, alleged to have followed a scratch from a foreign body, led to local folds of Descemet's membrane and precipitates on the back of the cornea, the aqueous beam was much intensified. After severe blows on the eye, without perforation, the aqueous beam may be very visible, with or without gross evidence of hyphema; or the evidence of hyphema may consist of no more than a trace of dust like deposit on the back of the mid lower peripheral part of the cornea, so fine that its color cannot be identified. Again, granular blood debris, doubtless corpuscles, may be present in the aqueous fluid of these cases in such profusion that, in the course of its circulation with the currents in the anterior chamber, it conveys the impression of falling down the back of the cornea to which none may be adherent.

In a boy in whom I saw this the day after he was hit in the eye by an air gun shot, which penetrated the conjunctiva but not the sclerotic, some blood was visible at a small point on the extreme visible periphery of the anterior face of the iris, as tho bleeding were occurring there from a rupture of the stroma. This was further suggested by the fact that the pupil margin, in the corresponding sector, was slightly flattened, owing to local paresis of the dilator fibers of that part. One hour after I had dilated the pupil with atropin (unfortunately I did not watch the eye during the interval) no bleeding point was to be seen on the iris and no granular material was visible in the aqueous fluid; suggesting that the dilation of the pupil had mechanically checked the bleeding from the iris; and that, in this short time, all the corpuscular debris had passed from the anterior chamber. The outstanding beam, however, remained very much intensified in this patient and is still slightly so four months after the accident.

A year ago a man came to me stating that, two days before, he had had a severe blow on the eye. The only evidence, by ordinary methods of examination, of injury to the eye was a slight mydriasis. The aqueous beam was, however, intensified and there was the faintest trace of a little fine faintly colored powdered debris, deposited in a vertical line—doubtless by the convection current—on the mid lower region of the deep face of the cornea. No granules were visible in the aqueous fluid. The visibility of the aqueous beam became normal two and a half weeks after the accident, but the slight mydriasis is permanent.

Some time ago I saw a patient who, when passing a cricket field, received a blow on the face with a cricket ball. He went to a doctor who "put something in his eye." The patient declared that his pupil was dilated before he went to the doctor; by a coincidence, the doctor died the day after, and information as to what was put into the eye was not available. The size of the pupil became equal to that of the other after a few days. The patient alleged that the vision of this eye was defective; it was. Both eyes were hypermetropic—the alleged injured one being the more so. But there was increase in the visibility of the aqueous beam. Subsequently pigmentary changes characteristic of concussion injury appeared at the macula, so that now the eye has very little sight that is of value. Yet just after the accident the only definite sign which, under the circumstances, indicated that the eye had been injured was the increased visibility of the outstanding beam of the aqueous fluid. The examination of many injured eyes has, however, shown me that the absence of this sign, in an eye alleged to have been recently injured, is no proof that the eye cannot have received a concussion injury.

When it is considered how the visibility of the aqueous beam of injured eyes may be increased, without any accompanying visible evidence of the presence of granular blood derivatives, the factors on which such increase depends are conjectural. I have seen this sign, very much in evidence, lasting some months in a case of hypotony following concussion injury to the eye. Mr. Treacher Collins suggested that effusion of serous fluid from the vessels of the iris due to the low tension, and consequent increase in the protein of the aqueous fluid, might be the cause of the increased visibility in this case. I might here point out that I have found long standing intensification of the aqueous beam in an eye which revealed, after a blow from a football, persistent increase of tension from causes which were not discoverable.

The factors on which the persistence of this sign depends—so that in some it disappears soon and in others it may still be seen long after the receipt of the injury—are not clear. It certainly is apt to persist for a long time in those cases in which evident inflammatory trouble follows the injury. I have noticed also that in other cases, e.g. after concussion injury, while the gross visibility of the beam may disappear in a comparatively short time, a slight pathologic intensification may remain long thereafter.

Occasional obscure affections of the conjunctiva are met with—I have seen three or four—in which evidence of associated intraocular trouble is revealed by increased visibility of the aqueous beam. One of these cases showed very fine debris adherent to the back of the cornea; one had no sign of keratic precipitates.

It is interesting to examine for this sign in cases of scleritis. Without now going into details, I would add that it is not present in all cases of scleritis, but it seems prone to appear in those cases in which the scleritis is tending to encroach on the periphery of the cornea. The increase in such cases may be present with or without fine keratic precipitates; it may precede the occurrence of precipitates. I have found very definite intensification of the beam in some cases of keratoconus.

The relationship of this sign to the various aspects of iridocyclitis is one for long discussion by itself; space prevents my entering upon it here. How far the manifestation of this sign is to be taken as indicative of the activity of a disease which is prone to run an irregular or relapsing course I am unable to say, even after long observations whose object was to arrive at some conclusion on this point. The sign may be present to only a slight degree—naturally difficult to see—in severe cases of interstitial keratitis; and when the attack has subsided the aqueous fluid may appear normal or may show only a very slight persistent intensification of its beam.

I believe that this is of clinical value in relation to the anticipating of sympathetic ophthalmitis; but I defer dis-

cussing this now before I may have had opportunity to make further observations.

There are certain not unfamiliar varieties of intraocular disease unobtrusive but serious, which manifest themselves chiefly by fine signs detectible, by ordinary ophthalmoscopic methods, in the posterior parts of the eye, e.g. fine dust like products profusely distributed thruout the vitreous, with or without retinal changes at the macula or elsewhere. Slit lamp examination of these cases may disclose the presence of fine changes at or behind the posterior lens capsule, and secondarily within the lens; and also in some, but not all of these cases, a few sparse and fine but definite deposits may be seen on the deep face of the cornea. But with or without the presence of corneal deposits these cases may reveal slightly increased visibility of the aqueous beam, which seems to be subject to variation with different states of the affection. Cases of this type, which begin with abundant vitreous "dust" as the most evident sign, may later come to exhibit fine but protuberant inflammatory exudations from the iris especially from the pupil border; but the slightly abnormal aqueous beam which such a case may reveal, does not appear to be especially evident, merely because it is associated with the occurrence of the visible iris exudation.

A case of luetic neuroretinitis in an old woman showed no sign in any part of the eye other than slight intensification of the aqueous beam; there was no deposit on the back of the cornea. A young woman who, from acquirement of the same infection, had dense vitreous "dust" in both eyes—yet no evident fundus lesion—revealed intensification of the aqueous beam definitely in the right eye, and scarcely, if at all, in the left eye; there were no keratic precipitates in the right eye, yet two definite round spots of moderate size were present on the back of the left cornea. A case of severe vitreous affection, with very large gross deposits, due to the same, but inherited, cause, showed this sign in the aqueous fluid only slightly. I did not, however, come upon the case until the disease had apparently for a long time ceased to be active.

In the Brit. Journ. of Ophthalmology for October, 1924 ("Microscopy of the Living Eye"), reference is made to the fact that in cases of simple detachment of the retina, the aqueous beam generally in the course of time manifests a very definite pathologic increase of visibility. Unfortunately the opportunities of examining such cases very soon after the detachment has occurred are not frequent, but, as far as I have been able to ascertain, the sign does not seem to appear until some time after the occurrence of the detachment. I watched twice weekly a patient who came to me just after the retina became detached—it seems four days after, from the history. The visibility of the aqueous beam began to increase a month after, and by the end of the second month it was pronounced. Another patient did not show intensification of the aqueous beam until two months or more after the detachment occurred. Cases of old standing simple detachment generally show a very definitely pronounced intensification, but I have seen two cases of two to three years standing in which the increased visibility, tho pathologic, was only slight; and I have come upon one case, that of a woman who is not a myope, in whom there is no pathologic increase in spite of a lasting detachment of the retina which occurred three years ago. It will therefore be seen that increased visibility of the aqueous beam, tho a usual sequel to simple detachment of the retina, is not necessarily a constant one; and it is a sign which does not manifest itself until a variable period after the occurrence of the detachment. Experience has led me to believe that the aqueous beam remains normal in cases of detached retina due to malignant growth; but obviously eyes are usually excised before sufficient interval may have elapsed for the sign to appear. However, I have not seen an intensification of the aqueous beam in any case of confirmed growth detachment, save in one instance in which it

was very faintly pathologic; and, of the cases I have examined, three (adults) had undoubtedly had affection of their sight during a period of not less than three to five months previously.

I ought to add that one case of a detachment situated rather far forward towards the ciliary region in the eye of an elderly woman, due, as I believe from ordinary clinical evidence, to an underlying growth, displayed a moderate pathologic intensification of the aqueous beam; but the patient disappeared and did not submit to the enucleation which was advised.

Admittedly trouble may have been present for a long time before advice is sought because of detachment of the retina occurring in the eye of a young child. I might, however, mention the cases of two children from each of whom an eye was excised because of detachment of the retina. Pathologic examination disclosed no growth under either of these detachments, but only inflammatory changes of the ciliary region and other parts. In both cases, clinical examination before excision had revealed very visible intensification of the aqueous beam.

I have watched for three years the eye of a young girl who, when advice was first sought, was examined by many ophthalmic surgeons. The case was a very unusual one, the main feature, besides a protuberance from the back of the iris, being a white membranous expansion which bore vessels over the posterior aspect of the lens. Various opinions were expressed, including the possibilities that it might be congenital, or inflammatory, or neoplastic, or a sequel to injury. The very increased visibility of the aqueous beam and the subsequent course of the case have shown that the affection was inflammatory.

I have found pathologic intensification of the aqueous beam, together with very fine dusty deposit on the back of the cornea, in a woman who had bilateral diabetic cataract.

It may be worth mentioning that certain cases of high myopia, more especially those whose fundi show myopic choroidal atrophy, sometimes have a slight intensification of the aqueous beam. In one patient it was more in evidence in the eye which was the more myopic. But the extent of the pathologic increase in such cases is very slight.

I have given, I hope, a fairly representative list of the types of cases in which this sign may be found. At present its chief clinical value would seem to lie in those cases in which its presence affords contributory evidence of injury to the eye; and also in certain types of inflammatory cases. Further investigation of large numbers of cases of the types I have referred to will be of interest, to determine the average period of onset and disappearance of the sign, as well as other considerations which will readily suggest themselves. It is clear that the value to be allotted to the sign is dependent on the experience of the observer. The very frequent examination of the normal aqueous beam preferably always with the same instrument, is the only way to acquire a sense of what is pathologic.

Some of the observations here quoted were made during a period of twelve months, from 1922 to 1923, in which I held, from the British Medical Association, a Research Scholarship for which I wish to acknowledge my indebtedness.

MARGINAL DYSTROPHY OF CORNEA: FURROW KERATITIS.

SANFORD R. GIFFORD, M. D.

OMAHA, NEBRASKA.

Seven cases of this condition are here reported, three of them observed from nine to eighteen years and others probably of long duration. The review of the literature is brought up to date and these points emphasized: The condition often begins in early life; not associated with arcus senilis; Rupture of cornea may occur spontaneously or with slight force; incisions in such corneas are dangerous and should be covered with conjunctival flap. The treatment is generally careful correction of the high inverse astigmatism. From the Department of Ophthalmology, University of Nebraska Medical College.

The opportunity to examine three cases of this somewhat rare condition with the slit lamp, and to go over the history of several other cases from Dr. Harold Gifford's case records seemed to provide material worthy of a brief report, especially as very little has appeared on the subject in the American or English literature.

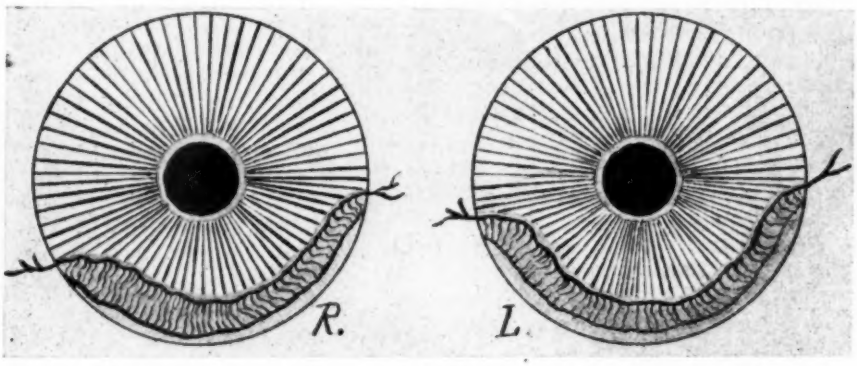

Fig. 1.—Appearance of eyes in Case 1 showing furrow near lower margin of each cornea.

Case 1. Mr. E., aged 29, was first seen by Dr. Harold Gifford in 1911. He complained of failure of vision in the right eye for the past 6 months. Vision was R. 20/100 and L. 20/20. Vision in the right eye was improved to 20/20-3 by −2.00 with −5.50 cylinder axis 90°. The left eye accepted +0.50 cylinder axis 90°. Both eyes showed a smooth groove near the lower margin a little to the nasal side. Each depressed area was about 2 mm. wide, smooth, not staining with fluorescein, and rising sharply at the central border to meet the normal corneal tissue, from which it was marked off by a fine grey line of opacity. Both peripheral and central to the groove were a few fine punctate opacities. There was slight pericorneal congestion, not especially marked at the site of the grooves, but somewhat more pronounced in the right eye, which also showed an enlarged pinguecula nasally. The eyes had been somewhat congested but not irritable or painful. The patient's general condition was excellent, and his history negative.

Eight months later there was little change in the patient's condition. Five years later the refraction had changed slightly, and was as follows:

R. +0.50⊂−5.50 cx. 70°. L. −0.25 ⊂+0.75 cx. 170°.

Both eyes now showed rather marked congestion, especially near the ends of the grooves, and many vessels were seen in the depressed portions. The groove was much deeper in the right eye, and an area of vascularized cornea with some punctate opacities was seen near the upper limbus. This condition was about the same one year later.

Six years later, or thirteen years after his first visit, the refraction was:

R. +1.00 with −5.50 cylinder axis 80° = 20/30. L. −0.50 with +1.00 cylinder axis 170° = 20/20.

The condition had not changed

materially. (See Figs. 1 and 2.) The central border of each groove showed a sharp greyish line of opacity. A large vessel at each angle of the grooves could be seen by the slit lamp to form numerous finer loops just under the epithelial covering of the groove which was intact. The exact curvature of the groove could be beautifully seen with the narrow bundle of light. The central border presented a sharp declivity, the peripheral a shallow slope. The posterior surface of the cornea showed no greater curvature at the site of the groove than that of the rest of the cornea, but the whole cornea could be seen to be much more sharply curved in the right eye. Treatment consisted simply in the correction of the astigmatism by glasses and a mild zinc collyrium.

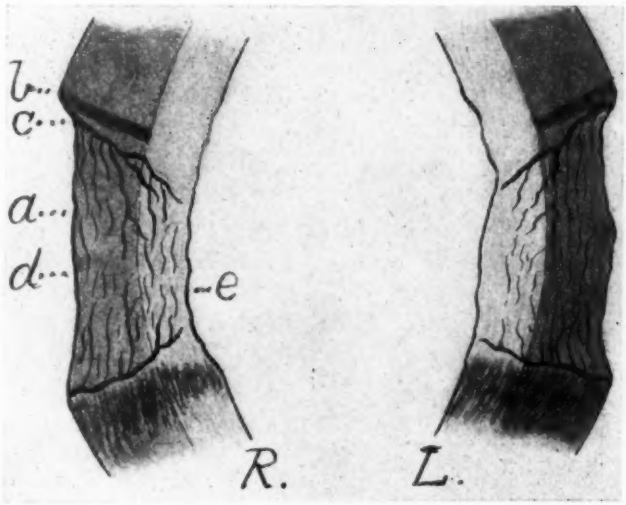

Fig. 2.—Represents section of groove and degenerated part of cornea in Case 1. b, grey line of opacity at central border of groove; c, sharp central border; a and d, degenerated portion of cornea showing new formed vessels; e, deeper layers of cornea unaltered.

Case 2. Mr. W., aged 33, was first seen by Dr. Harold Gifford in 1907. His case is interesting as it showed the complete process from the earliest beginnings. At his first visit the patient complained of recurring attacks of inflammation in each eye. The right eye showed a patch of congested conjunctiva at the outer side, some of the vessels extending into the cornea. There were peripheral corneal opacities, which were thought to be the scars of former ulcers. Vision was normal in each eye without correction. A year later the localized congestion previously seen was more marked. The left cornea was seen to have an opaque area at the temporal margin, and was now slightly myopic, requiring —1.00 with —0.25 cylinder axis 90° to give 20/20 vision. It was not until one and one half years later that the typical groove formation was seen, which explained the nature of the case. The groove was now definite at the upper margin in each eye, tho deeper in the right. The left cornea seemed to be more prominent than normal below the groove. Attacks of inflammation had occurred several times since the last visit. Vision was now R. 20/100, L. 20/50. Refraction showed: R. —6.00 cx 75° = 20/30-3. L. —0.75 ⊃—1.50 cx 65° =20/20.

No constitutional cause for the degeneration could be found. Two years later the right eye showed a ring of faint opacities, ⅛ inch wide all around the cornea. Peripheral to this was a clearer zone of cornea, with fine vessels crossing it. The cornea rises sharply beyond the central line of opacities and a more diffuse opacity extends central from this border. At one point, up and in, this opacity reaches half way to the center of the cornea. The left eye shows much the

same condition; the cornea being more vascular in the depressed portion, but the change in curvature not being so marked as in the right eye. Refraction was:

R. +2.00⌒ —11.00 cylinder axis 25°. L. —3.00 cylinder axis 65°.

Two years later the condition was much the same but the astigmatism had increased.

R. +3.00⌒—14.00 cylinder axis 75°. L. —4.00 cylinder axis 55.

terior surface of the cornea showed irregularities of curvature at the site of the grooves. This was more marked in the right eye, and evidently represents the beginning of a localized ectasia. Thus it might be supposed the further ectasia would occur in this case, while in Case 1 the condition seems relatively stationary, and localized ectasia may not ever occur. It seems worth while to distinguish localized ectasia, the protrusion of the

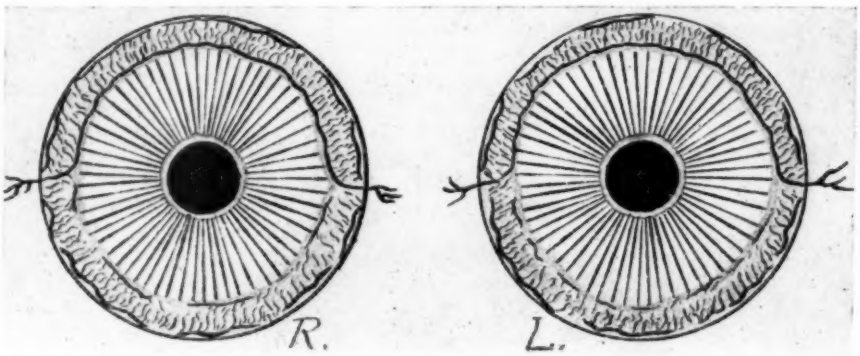

Fig. 3.—Case 2 groove extending around cornea near margin.

After another two years refraction was:

R. +3.00⌒ —16.00 cylinder axis 80° =20/20. L. +1.00⌒ —7 cylinder axis 70° = 20/20.

Six years later in 1924, or 17 years after his first visit, refraction was:

R. +1.00⌒ —16 cylinder axis 70°= 20/40. L. —8.00 cylinder axis 65°= 20/30.

The condition is apparently much the same as that observed ten years before, except for the increase in astigmatism. The discrete opacities have been included in the thinned area of the cornea and the prominence of the central portions has slowly increased. (See Figs. 3 and 4.) The only treatment was yellow oxid ointment and a zinc collyrium which latter seemed to help in lessening the severity of the congestion and irritation.

The slit lamp showed the exact distribution of the finer vessels, which all stopped sharply at the grey line. They were present in the entire thickness of the thinned area. With the small beam of light the findings differed from those in Case 1 in that the posthinned cornea at the site of the former groove above the surrounding cornea, as described in many cases, from the general ectasia of the whole upper half of the cornea or the whole cornea, which occurs in all cases, as shown by the high inverse astigmatism. Localized ectasia was present in only one of the present cases (Case 6), except in the slight degree in Case 2, as seen with the small bundle of light.

Case 3. Mr. W., aged 32, was seen by Dr. H. Gifford in 1915. At the age of 18, the left eye had an attack of acute inflammation, lasting three days. Ever since it has had similar, but less marked attacks with free intervals of two to three months. In recent years the attacks have been less frequent. For the last two years vision has been failing in this eye. The right eye has always seemed normal. Vision is R. 20/20, L. 20/200. Refraction shows:

R.+0.25 c.ax. 95°=20/20. L.+1.50 with —2.75 cyl. ax. 70° = 20/30-1.

The left eye shows a shallow groove around the upper half of the cornea. This is separated from the normal cornea by a sharp line of opacity which

in places separates to two lines. The depressed area is relatively clear except for numerous fine vessels passing across it, most arising from a larger group of vessels at the temporal end of the groove. One year later the refraction and appearance of the eye was about the same. The right eye showed no sign of a similar trouble.

Case 4. Mrs. A., age 35, was seen in October, 1919, on account of poor vision in the right eye, worse for the last three weeks. The right eye showed a typical furrow just inside the lower limbus, including the lower half of the cornea with a few fine opacities near the central border. The left eye, which had never given any symptoms, showed a narrow but sharply defined furrow at the upper limbus. The right eye showed slight congestion in the region of the furrow, the left none at all. Vision with correction was 20/15 in each eye. No symptoms had been complained of in the left eye. These furrows were similar in every way to the case previously described. The patient has not returned for further observation.

Case 5. Mr. L., age 51. In this case, both eyes were affected with a typical furrow formation, which in the right eye extended two-thirds way to the pupillary margin, with an area of diffuse opacities extending still further toward the pupil, so that almost the whole upper half of the cornea was opaque. In the left eye a very much narrower furrow was present. The refraction was:

R. $-2.50 \frown +1.75$ cyl. av. $160° =$ 20/20. L. $+1.00 \frown +2.00$ cyl. ax. $170°$ $=$ 20/20.

An interesting point about this case was the presence of distinct scars of diffuse trachoma for which he had received treatment when 20 years of age. Thus a great deal of the opacity in the right eye was probably due to pannus and the furrow may have also developed as a result of it. The slit lamp, while showing the exact configuration of the furrow in the anterior surface, revealed nothing as to the curvature of the posterior surface which was concealed by the opacities. The patient had worn glasses for 23 years, and it is quite likely that the dystrophy had been present for that length of time.

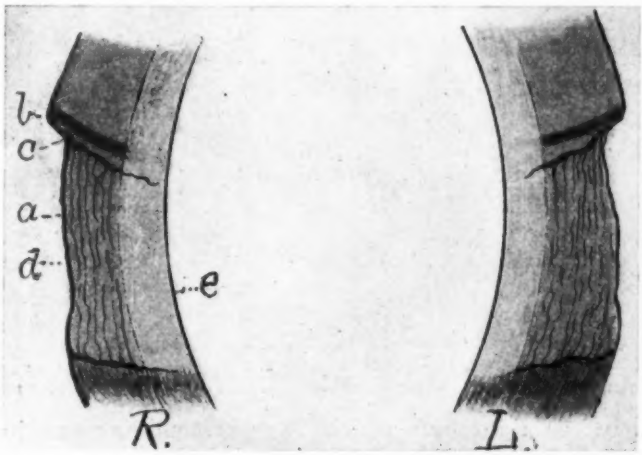

Fig. 4. Represents sections thru the cornea in region affected. b, line of opacity at central border; c, abrupt edge of groove; a and d, depressed, degenerated portion of cornea with new formed vessels; e, deep layers less altered.

Case 6. Mr. H., age 87, is the only case showing the advanced stage of the dystrophy, with typical marginal ectasia about 2 mm. in diameter, extending around practically the whole upper part of the left cornea. The eye also had a mature senile cataract which was operated on by Dr. Harold Gifford, who will report it in detail elsewhere. By using trichloracetic acid on the upper portion of the cornea, which was paper thin, and covering it with a sliding conjunctival flap

from above, a good operative result was obtained (V 20/70 which could be improved by a discission). The right eye had been lost, following a cataract operation two years before.

This case is interesting in view of Axenfeld's experience, to be mentioned later. Axenfeld suggested the use of the conjunctival flap, if he should have to operate on such a case again. It seems very likely that in this case, marginal ectasia had also been present in the right eye, which was probably operated on without the precaution of the flap, with the resulting loss of the eye, due to the extreme thinness of the cornea, which proved insufficient for firm healing.

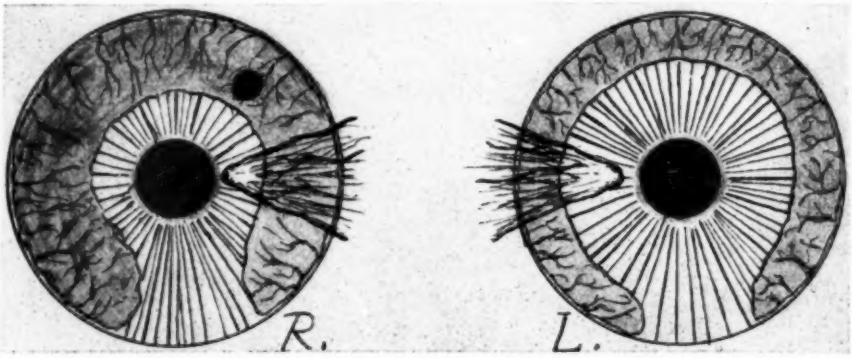

Fig. 5. Condition in Case 5, a very advanced stage of corneal change. A part of each corneal margin below remains unaffected. Right eye shows small prolapse of iris in upper nasal region.

Case 7. Mr. S., age 84, was only seen once but showed a very advanced stage of the condition. He was 20 years old when the right eye was injured, after which the sight had remained poor until 20 years ago, when he had become entirely blind. The left eye had been failing for 5 years. The right eye was found to have no light perception, the left saw hand movements in the temporal field only. Besides the corneal condition the right eye showed a complete cataract, with a small button of iris caught in the cornea up and in and covered by epithelium only. The left lens showed fairly dense opacities in the posterior cortex, which prevented a view of the fundus. The tension was normal. Most of the right cornea was involved in a vascularized horseshoe shaped opacity, enclosing a clear area of about one-third the size of the cornea, connected to the limbus below by a bridge of clear cornea. (See Fig. 5.) The opaque ring was depressed considerably, and separated by a distinct line from the clear area, which appeared to be elevated. The left eye showed the same condition, the horseshoe shaped opacity being almost exactly symmetric, but not so wide as in the right eye, leaving a larger clear area. Evidently the condition was of many years standing; and in the right eye the thinned cornea had perforated, the hole being stopped by iris. It is possible that this may even have occurred at the time of his injury 60 years before, tho some slight trauma may have caused it later. What complicating conditions caused the loss of sight is a matter of conjecture.

LITERATURE.

There comes to hand at this moment, the excellent report on this subject by Gonzalez, in the June, 1924 number of this journal (v. 7 p. 452). It includes a good bibliography and resumé of the literature, so only a few points not noted by him will be emphasized, and those references added which complete his bibliography. His paper is the most complete one in the American literature, and I have found only one good one, that of Fisher, with pathologic examination and discussion by Coats, in the English literature. Descriptions of 52 cases were found in the literature, the present 47 cases making a total of 559. The majority

have been seen in persons over 45, and some observers have considered the condition a senile change related to arcus senilis (Fuchs, Gilbert). This view cannot now be held, however, as a fair number of cases in younger persons are on record as pointed out by Gonzalez. In his case the condition probably began at 16, and in Marquez' case at 10 years of age. Of the present cases 4 were under 36, with no signs of arcus.

In many reported cases, it is likely that the condition was of long standing when first seen. Fuchs recognized this form of degeneration as occurring rarely in younger persons; but thought, on account of its common location above, where the gerontoxon commonly develops first, that if not actually a result of arcus, it represented a degenerative process analogous to it. Handmann has even suggested that the flattening of the vertical meridian which is so frequent in old age represents a tendency toward the same form of degeneration or weakening of the upper portions of the cornea. In any case, the upper margin would seem to be a place of least resistance in the cornea; making it early susceptible to both marginal dystrophy and gerontoxon, even tho these be entirely different conditions. There can be no doubt that the condition is degenerative in origin, the inflammatory symptoms observed in about half the cases being secondary, and possibly due to cellular necrosis in the cornea. The small, discrete, greyish or yellowish opacities, observed in the present cases and in many others, possibly represent such areas of necrosis or deposits of degenerative products in the cornea. In line with its dystrophic nature is the usual involvement of both eyes.

Besides Gonzalez's case, and the four mentioned by him, only those of Vavouline, one of Früchte, and 2 of Gilbert have been found which were monocular. To this may be added Case 3 of the present report. Gonzalez states that the male sex is almost exclusively affected. He mentions Zentmayer's as the only case reported in a woman. I have found Brewerton's case, one of Lauber's, 2 of Gilbert's, 3 of Fuchs', 2 of Axenfeld's and one each of Fisher, Knapp and Markus, which were also in women. One of the present series was a woman, six were men. This still leaves an enormous preponderance of men (about 75%). This would probably point to exposure or heavy labor as being an etiologic factor.

Among the complications of the condition, the most dramatic in its interest is rupture of the cornea, spontaneously or due to some slight trauma. Uhthoff's patient, a man of 59, had noted attacks of inflammation in each eye for 10 years. His first visit was after he had struck his right eye with his little finger, producing a rupture at the thinned upper margin, thru which the lens had escaped. The typical marginal dystrophy with groove formation was seen in other parts of the cornea, and in the other eye. Twelve years later he came in with an exactly similar condition of the left eye, which he had struck with his thumb while washing. He was left with vision of counting fingers in each eye. Uhthoff had seen one other case with perforation and iris prolapse. Seefelder saw two cases with perforation, in the grooved area, after being struck, one by a piece of wood, the other by a child's hand. In Schutz's case the perforation with prolapse of iris was spontaneous, as was also true in the case of Drüner and Wiedersheim.

Another possible complication is secondary infection of the thinned area. Axenfeld's case developed typical Mooren's ulcers, at the site of an old marginal dystrophy in both eyes, the resulting opacity involving the whole right and the upper half of the left cornea.

In a second case Axenfeld was obliged to operate for cataract on a woman with bilateral marginal dystrophy with ectasia. The borders of the operative wound became infiltrated and a ring ulcer developed, which healed, however, leaving vision of 6/12. He believed this process to be a form of necrosis, due to poor nutrition, and thinks the case is evidence that faulty

nutrition is a cause of marginal dystrophy.

Rupprecht's case developed a serpent ulcer, and the eye was enucleated. Sections showed complete absence of Bowman's membrane, and almost complete absence of corneal lamellae in the ectatic portion. The process had involved the whole thickness of the cornea, and aqueous had been forced into the resulting connective tissue, forming a cyst. The complete absence of Bowman's membrane over the diseased area agrees with the findings in Seefelder's and Fuch's cases which also came to enucleation. The same degeneration of corneal lamellae was also present in these cases, extending entirely thru the cornea in Seefelder's case, but only involving the superficial half in Fuchs' case, the loss being replaced by granulation tissue which in Seefelder's case, contained numerous fat globules. Much fat was also present in Kuriyama's case, these four cases being the only ones in which a pathologic examination of the whole eye was made. The portion of cornea excised by Fisher and examined by Coats, showed a most unusual thickening of Descemet's membrane, five or six layers being distinguishable, giving it a total thickness of 55 microns in one place. This thickening was evidently formed by successive deposits, to cover numerous cracks, which were seen to be present in the older layers. Trachoma with pannus was present in the cases of Ischreyt and Rshanizyn, and was thought to be the cause of the degeneration.

The myopic astigmatism, present in nearly all cases, may reach extreme degrees. It reached 21 D. in Marquez' case, 22 D. in that of Ischreyt, and fairly often 16 to 18 D., as in my second case. As careful correction by glasses is the only form of treatment of value in most of these cases, they should be refracted carefully and the refraction repeated whenever further loss of vision is noted. The increase in astigmatism, while progressive up to a certain point, often stops; leaving refraction about the same for years as in the first case of this report. The condition is often noted only when the change of refraction requires correction, and marginal dystrophy should be thought of in any case showing a high degree of inverse astigmatism, especially if this increases rapidly. Probably many cases are missed entirely, being considered merely as high astigmatism, the furrow, concealed beneath the upper lid, never being seen at all.

In the United States, the only cases previously reported have been those of Zentmayer, Knapp and Schutz. Gonzalez' case is reported from Mexico; and Brewerton, Markus, Collins, Fisher and Coats have apparently reported the only English cases. Cases are probably as frequent here as in Germany or France but are either not recognized or not reported. It is worth noting that Frank described four cases of this disease in a dissertation in 1896, or four years before Terrien's report.

The possibilities of treatment are confined to careful correction of the refractive error and a mild antiseptic collyrium for constant use, to protect against secondary infection. Many cases, like Case 1 of this report, remain stationary for years on such treatment, with practically normal vision. Cauterization, as suggested by Terrien, will be reserved for cases in which the ectasia is certainly progressing, and would have to be performed very carefully. With the possibility of perforation in mind, patients should avoid heavy work where possible; and constantly wear glasses with heavy frames as the best protection against slight traumata.

Where a cataract operation must be done on a patient with this condition, the use of trichloracetic acid on the thinned area, which is then covered with a sliding conjunctival flap, would seem to promote healing and guard against rupture of the ectatic cornea.

The author takes this opportunity to thank Mr. Pelikan and Dr. W. A. Cassidy for their kindness in making drawings from his sketches.

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ETIOLOGY OF GLAUCOMA.
OPERATIONS FOR THE RELIEF OF TENSION.

H. Dickson Bruns, M. D.

NEW ORLEANS, LA.

This paper emphasizes the occurrence of two distinct forms of glaucoma; the congestive, or paroxysmal type, and simple glaucoma, the latter associated with elasticity of the sclera. Eyes examined microscopically present the changes of the late stages. Etiology will be shown only by early clinical studies. Simple glaucoma is almost as common as the other forms, being especially frequent among negroes. Operation is the essential treatment for glaucoma. Miotics are generally useful only as a temporary resource. The writer has done only iridectomy and trephining.

An invitation to discuss a paper by my friend, Dr. J. W. Jervey of Greenville, South Carolina, has led me to rearrange my ideas upon this subject. I suppose we must all do it now and then in the course of a long professional lifetime; but it is with sincere diffidence that I present mine. My excuse is that "A hair perhaps divides the false from true" and while we are awaiting the flash of inspiration that may give the key of the mystery to some superior mind, our hope is to turn and twist the matter again and again, tearing out of our imperfect theories all that time proves false, inserting better material and learning from the gaps we leave the knowledge we stand most in need of.

On reading Dr. Jervey's paper (Southern Medical Journal 1924, v. 17, p. 218), I was struck at once by the way he turns his back upon the retention theory without so much as a word of consideration. Is this an indication that the minds of most ophthalmologists are now swinging back to the old secretion theory, the theory under one or another of its forms of Donders and von Graefe? I believe that most of us have returned to von Graefe's view, that all the symptoms of glaucoma are to be ascribed to continued abnormal elevation of tension. All that I have to say rests on that assumption.

Almost, it follows as a consequence, that we can no more hope to distinguish the true cause of glaucoma by fixing attention on the symptoms of an established case than, as has been well said, we can by dissection of the long diseased specimens, which find their way to the pathologic laboratory. The chain of causes and effects has become too entangled. It

is, then, upon the incipient cases, that we must concentrate our observation and our reasoning.

The well known fact that blocking of the anterior chamber produces elevation of the intraocular tension is the foundation of the retention theory.

We need not stop to consider the extreme instances of blocking, moving forward of the lens and iris, adhesion of the root of the iris to the cornea, etc., found by dissection, for I believe we are all now fairly well satisfied that these are the consequences of continued high tension. So that the formula has been changed, and we are asked to see in shallowness of the chamber and narrowing of the angle, with impairment of filtration, the cause of the abnormal tension.

Let us pass over the commonplace that many middle aged and nearly all old people have shallow anterior chambers and narrow angles, and that only a small proportion ever develop glaucoma. Taking due care, we are often obliged to dilate the pupils of old persons with the result only very rarely of precipitating a glaucomatous attack. The last case in which the writer had the misfortune to precipitate an unexpected attack of glaucoma, was in a middle aged man with a bright blue iris, and nothing about the pupil or depth of the anterior chamber to attract attention. He cannot refrain from saying that he does not consider the mydriatic test a legitimate diagnostic method, having seen one case at least in which a deplorable condition resulted. He has more than once dilated the pupil in simple glaucoma without any untoward effect.

We must turn to the incipient condition, "the prodromal or irritative attack," for any real evidence for or against the retention theory. Tho I have seen few patients during such an attack, I have the impression that in one at least the anterior chamber was not strikingly shallower than that of the fellow eye, or of less than the average depth in persons of the same age. If interference with drainage from the anterior chamber be the prime cause of the increase of tension, should we not expect to see some deepening of the chamber, at the very beginning of a first prodromal attack? In certain cases of acute uveitis we may see in the beginning increased depth of the chamber coexisting with increased tension. Should not the slow accumulation of aqueous in a chamber with impaired drainage produce the same effect as the accumulation of aqueous more rapidly than the normal drainage apparatus can carry it off i.e.—deepening of the chamber?

This question of the relative depth of the chamber during a prodromal attack would appear to be an important point for, or against, the retention theory. I hope that colleagues of wider experience can bring forward precise observations; and it might be suggested that every one who sees a patient in one of these attacks should make careful notes, not only of the condition, but of the changes taking place during the attack, the state of the fellow eye, the age of the person and the number of the attacks. If such exact information is to be had, I do not know where to find it. Maybe this is one of the gaps in our knowledge which I mentioned. Perhaps the perfection of anterior chamber goniometers will end all doubts.

Granting that more or less obstruction of the anterior drainage area is the cause of glaucomatous tension, the question as to the nature of the obstruction arises at once. Clearly it must be of a kind that can temporarily subside or be overcome. For we know the prodromal attack after a longer or shorter duration passes off to be renewed once again, or oftener, until, as we say, glaucoma is established. I confess I cannot conceive of any diseased state of such structures as the pectinate ligament and Schlemm's canal that might wax and wane in the manner necessary to fulfil these conditions. It is needless to repeat that changes found in eyes long affected are beside the mark. We can readily imagine that more or less solid material might accumulate in the drainage areas and obstruct them; but it is hard to extend this to the conception of an obstruction large or solid enough to produce the phenomena of the

prodromal attack, and yet capable of being so completely swept away as to permit of the organ regaining its normal functions; and especially if this may occur more than once.

If, however, the obstruction in the angle must be considered as permanent, one supposition to account for the subsidence of the prodromal attack is, that the increased pressure must finally force the intraocular fluid thru those posterior drainage channels about which we know so little. It is to be observed that the stoppage could not from the first have been complete, if the chamber becomes shallower, for the lens could only advance after a certain portion of the aqueous had been evacuated from the chamber.

The fact that after the injection of the anterior chamber with a suspension of india ink the particles may be found in the episcleral veins, tho it has been denied that this can take place under normal pressure, suggests that the stoppage of these spaces by solid particles is difficult.

This brings us to a consideration of the Secretion Theory, which postulates an abnormal increase in the amount of the intraocular fluid as the first event in the process resulting in glaucomatous tension. A belief in a current in the intraocular fluid is necessary to the secretion, but not to the retention theory.

Belief in an outpouring and an escape of the intraocular fluid, so slow as just to fall short of stasis, is compatible with the retention theory. We have no knowledge of the time needed for obstruction of the filtration apparatus to reach the degree requisite to the production of a prodromic attack or an acute outburst. While the existence of a current in the intraocular fluid cannot yet be said to have been demonstrated beyond the peradventure of a doubt, the weight of evidence seems to be decidedly in its favor. This movement in the fluid must, I believe, be in the main the product of two factors, the pressure under which the fluid escapes into the eyeball and the elastic resistance of its capsule.

In the normal eye I can only imagine that the effect of these factors constantly and momentarily fluctuates. At one instant more fluid enters the eye than it contained the instant before. Immediately the elastic capsule, by that much overdistended, squeezes it out of the normal points of escape.

In this way the proper tension constantly swings to and fro across the line we call a norm. It is certain that the natural and wonted circulation of blood thru the organ is necessary, among other things, to the maintenance of this norm; and it is in agreement with our physiologic knowledge that an increase in the supply of blood to the organ, an elevation of the local blood pressure, will have as one of its effects an increase in the amount of intraocular fluid entering the eyeball.

When this happens we shall have a rise of tension, and if the extra fluid be considerable in amount, or if the capsule cannot promptly squeeze it out, or if both conditions exist at the same time, the rise may be sufficient to cause a prodromic attack, which will last until the contracting capsule rids the eyeball of the extra fluid.

Not only will the repeated unusual overdistention cause some loss of elasticity, but we know that the sclerotic, like all the connective tissues, loses this property progressively with advancing age. Therefore the time required for restoration of the normal tension will be considerably increased by this senile loss of elasticity. This function of the sclerotic, I think, must be reckoned with in forming any plausible theory of glaucoma.

The blood vessels, of course, are controlled by nerves and the state of local blood supply and pressure is regulated by the central nervous system. Causes acting thru that system, shock, strong emotions, fatigue, etc., usually precede, and we must suppose, excite prodromic and acute attacks. This is everywhere attested by our literature. In this connection it is worthy of note that the blood vessels of the face are in civilized man most susceptible to the emotions, as in flushing, rushes of blood to the head, etc.

It is one of the merits of the secretion theory that it takes account of

this admitted influence of the nervous system.

And behind this, what? Must we suppose some disturbance of nutrition; something added to or subtracted from the pabulum of the nerve center peculiar to the glaucomatous? The temptation to fancy such a state of things lies, of course, in the involvement of both eyes in primary glaucoma. If the patient lives long enough, the second eye is certain to be attacked. If this be so, we probably have long to wait upon others, the organic chemist, the experimental physiologist, for the solution of our problem. We now only touch the hem of such knowledge. But, tho it may, like the siren's song, be "a difficult question," it is not beyond the reach of ingenuity, as the discovery of the vitamins should assure us. On the other hand we may take the view that both eyes are in every way so absolutely identical that whatever determines the occurrence of glaucoma in the one will lead also to its appearance in the other.

All that I have said refers solely to the congestive type of the disease. When we come to consider the non-congestive type—simple primary glaucoma—we have to abandon the idea that narrowing of the drainage angle, or a degree of obstruction of the Fontana spaces, is a necessary starting point in bringing about rise of tension; for in these cases we see anterior chambers not shallower than common at the patient's age, and sometimes even apparently deeper. Yet I cannot see how any one watching a "simple" case developing one after another the same symptoms that we see in the congestive type and finally, tho more quietly and slowly, coming to the same bad end, can doubt that it is the same affection.

Dr. de Schweinitz in his well known text book in describing glaucoma simplex, the tersest and the best description I know of, expresses the opinion that this is now the prevailing view among ophthalmologists. As an adequate idea of what the writer means by glaucoma simplex is necessary to the clarity of the argument, and as an examination of the literature will show, I believe, that it has not generally been treated with either the fullness or the minute exactness of the other type, permit me a brief digression.

We constantly see articles describing forms of treatment or the results of operations in which from first to last it is never made clear what type of disease is under consideration. Simple glaucoma occurs in my practice, as often, or almost as often, as the congestive type. The negro, as I see him in our clinics, seems to be especially liable to simple glaucoma, and to present the disorder in a particularly pure uncomplicated form. Therefore I have sat down before it in a receptive state of mind for many years.

In the earliest stage, there is nothing to attract our attention to the eye, and our patients do not apply to us for relief of any symptom. The condition is only discovered by the routine examination with the ophthalmoscope, during measurement of the refraction.

There is now under my care a woman of good social position and intelligence, age 48, in whom the discovery was made during measurement of the refraction: R., glass, = 20/20; L., glass, = 20/15, some letters. Schiötz, average of 3 readings with different weights, R. 26.6. L. 21.6.

Both corneas are anesthetic; both discs excavated, but R. deeper with beginning break of continuity in vessels. R. field of vision all the upper —inner half gone, to within 5° of fixation point. L. normal. Pupils small and equal. Anterior chambers quite normal for her age.

A little later our attention may be caught by a small difference in the size of the pupils and a slight conspicuousness of the penetrating veins of the more affected eye. But, as Morax says, unless we are haunted by the fear of a mistake, even these trifling signs can be readily overlooked.

The negro does not usually come into the clinic until one eye is very badly damaged and the other failing, and even then seldom with a complaint of loss of vision, but of some-

thing "wrong with his sight," which is a vain attempt to describe a contracting field. An ignorant field hand, however, once made her difficulty clear to me by the inspired description, "I see like I was lookin' thru a knot hole."

When, after a long course, sight has been lost, the veins become more conspicuous, the tension much higher, the anterior chamber shallower, and often such severe pain sets in as to demand relief by operation. Finally, the condition is that of absolute glaucoma, and hyperemia accompanying changes of degeneration appears; but even then the excessive dilatation of the pupil and the marked dullness of the cornea seen in the hyperemic type seem to be lacking.

Up to this, the corneas are bright. Often, but not always they are anesthetic, and this frequently only in spots. The pupil grows wider and wider, but is easily contracted by miotics. Good central vision and good color perception persist for years, often together with damage to the field that amazes us. The "knot-hole" woman with a field nowhere exceeding 5° had not noticed change in her sight.

The disease is always more advanced in one eye than the other, usually much more so, as again illustrated by the case given. Cupping of the disc appears early and presents the characteristics seen in the congestive type. Tension is at first low compared to that found in congestive cases with similar changes in the nerve head and the field of vision. But it rises steadily with the progress of the disease, and often reaches a high degree, over 60 Schiötz by the time the vision is lost, and before degenerative changes occur.

I have never measured the tension of a patient more than once during a day, but I have never on any later day seen the tension fall below that first noted, unless a miotic was instilled. We should not be too much misled by these low tensions. The more we have used the tonometer the more convinced we have become, that what we must regard as normal tension, as indicated by this instrument, varies greatly in different individuals.

It would seem axiomatic that the tension of a pair of normal eyes must be equal. When therefore, we assure ourselves by repeated measurements that, while the tension of both eyes of an individual is what we regard as low, there is a marked difference in this respect between them, we are justified in concluding that there is something wrong with one at least. The sum of the symptoms, let alone the course of the malady, will enable us to judge which is affected, or we may find both involved, but one much the more. There are cases in our hospital records in which the tension of one eye is decidedly lower and the difference decidedly greater, e.g., 17 and 33; 18 and 35, than in the case cited. Indeed the difference is usually great. While perfect equality of tension may exist, it is so rare as to call for especial investigation. This difference in the tension of the two eyes seems to me one of the most valuable indications given by the tonometer and from the little I know does not seem to have been sufficiently emphasized.

Finally, altho I know the weight of authority is heavy against me, I am at present persuaded that the simple never becomes, never changes into the congestive type; a belief fostered by having observed many cases of the simple type, especially in negroes, from one inspection, to many during the course of years. Nor have I ever seen a case showing the one type in one and the other in the fellow eye.

I do not now believe that the simple ever warns of its approach by the prodromic attack seen in the congestive type, with its steamy cornea and consequent rainbow like halos. No patient with the earmarks of the simple type, comparatively low tension together with cupping of the disc, impaired field, but good central V. and color perception, have ever volunteered to me a complaint of seeing rainbows. Nor have I been able to elicit such a complaint, leading questions, of course being avoided.

In the simple type, then, it is evident clogging of the anterior drainage

area plays no part in starting a series of symptoms exactly parallel to those of the congestive type; the hyperemia and the high tension only being conspicuous by absence. Holding to the belief that in both types the phenomena are the result of the abnormal tension, clearly some condition is added in the one, or lacking in the other. That condition I believe to be the presence of a relatively high degree of elasticity of the sclerotic coat in the simple, and the lack of it in the hyperemic type. Granting this, we can readily imagine, that when one of the disturbances of circulation common to the glaucomatous occurs in a person whose sclera remains elastic, enough expansion of the globe takes place to compensate for the increase in the amount of the intraocular fluid. The elastic recoil begins at once to squeeze the excess out at the drainage openings, and there is no prodromal attack. We see an analogous process in congenital buphthalmos, but here the anterior drainage apparatus being absent, we believe, the distention goes on until the excessive stretching leads to disorganization, or some slight accidental traumatism causes rupture. It is in the normally *soft eyed* persons, as shown by the low tension at first in the better eye, that the tonometer has made known to us, that we suppose glaucoma simplex to occur.

I think it likely, too, that the simple type begins at an earlier age than the hyperemic. The youngest person with the disease I ever saw was a girl of 15 with glaucoma simplex in both eyes. It is, however, extremely difficult to determine this, for the disease is so insidious, good central vision is so long retained, that years may elapse before the second eye is so much impaired, as to attract the patient's notice and cause him to consult the surgeon. Dr. C. E. Granberry, the resident surgeon attached to my service at the Eye, Ear, Nose and Throat Hospital, was kind enough to tabulate the ages and the tension of patients with this type of the disease, admitted to the clinic during the past five years. The figures did not confirm my impression; I think, because most of the patients did not come until the second eye became gravely involved. There were 53 cases with an average age of 52+ years. The youngest case was 15 (mentioned above), but there were, besides, 6 between 35 and 40 years; the oldest was 74.

This slow progress is well illustrated by a white woman, 67 years old, upon whose R. E. I did an Elliot trephining nine years ago. At that time, T. (Schiötz) was R. 35 mm. and L. 35 mm. Now T. = —1., R. E., has good filtration, but is blinded by a mature cataract. L. E., altho there is a pronounced cup, shows no contraction of the field or scotomata. The T. (Schiötz) = 32½ mm. and V. only 20/XL, as there is slight cloudiness of the lens. There is slight enlargement of L. blind spot.

That cupping of the disc should occur early, while tension is still comparatively low, is naturally explained by supposing the lamina cribrosa to be especially yielding in individuals whose scleras retain a considerable degree of elasticity.

On the other hand it is hard to see why such profound changes can be wrought in the field of vision by tensions relatively so low. I can only fancy that we have been misled as to the consequences in this delicate organ of any rise above the normal tension, by the more constant fixation of our attention upon the higher tensions of the congestive type. We know that the unmitigated pressure of the acute congestive attack can be tolerated for a fortnight, say, without evil effects, while the damage done by the much lower long continued tension of the mild chronic congestive variety increases steadily with the passage of time. It seems possible that an eye congenitally adapted to the enjoyment of health under a certain tension, always suffers if that tension be long increased, even by what we are accustomed to regard as a slight amount. Perhaps, too, the early cupping of the disc leads to a sustained and injurious pressure of the nerve fibers against the rim of the scleral opening.

That the sclera, like some other sub-

stances, loses in elasticity by frequent or continuous exercise of the property, and that the amount of intraocular fluid poured out increases with repetition of the vascular disturbances, seems not improbable. Beyond a doubt, we see the anterior chamber grow shallower as the disease progresses, while the tension becomes higher, approaching towards the end the degree met with in the congestive type.

Finally, it appears not only possible, but probable, that both obstruction of the drainage apparatus and increase in the amount of intraocular fluid play a varying part in the mechanics of glaucoma, for it is really the mechanics rather than the etiology we have been considering. In all cases the lens eventually moves forward, soon in some, late in others, to a greater or less degree; and the preponderance of obstruction on the one hand, or of vascular derangement and increase of secretion on the other, together with the degree of elasticity of the sclera, determines the type, and in the congestive type the variety displayed.

OPERATIONS.

My experience with operations for the relief of glaucoma is restricted to two kinds.

I owe an apology to Dr. Jervey. Circumstances have precluded a trial of his operation during the past winter. Up to the present I have not felt myself justified in doing any of the decompression operations but the classic iridectomy and the Elliot trephining operation, save simple sclerotomy in glaucoma simplex.

Let me say at once that I have lately done sclerotomy in two favorable cases, making the section one and a half to two millimeters behind the limbus corneae, with complete disappointment. The effect on tension was temporary, lasting weeks or months, miotics having again to be resorted to. Any operation in which this is necessary I consider a failure; we have but complicated our treatment and exposed our patient to an additional danger. I shall do trephining in one of these cases at the earliest opportunity. This I believe to be the operation of choice in the simple type. The danger of late infection seems to have been exaggerated and can be lessened by teaching the patient to wash out his eyes freely night and morning with a simple solution of borax.

I have more than once noted the hypotension after trephining, to which Dr. Arnold Knapp first called attention, I believe. I observed it most markedly in the case of a white woman of 56 both of whose eyes had been trephined by another surgeon. T. was R. E. —2. L. E. —2. Field of vision, R. contracted; L. inside of 10°; V. R. = 20/20. L. = 20/70, with correction. She had become alarmed by an edema of the whole bulbar conjunctiva completely surrounding the cornea of R. E., and which I estimate, must have had a depth of about 2 mm. It gave the look of a glass eye. Up to this time I have never seen any bad consequences result from this hypotension, but I view it with anxiety. Indeed I have rarely seen anything but benefit follow trephining, and it affords great relief to the pain which attacks hopelessly blind eyes in the last stages of the simple, as well as the congestive type. I am glad to be able to confirm the experience, recorded years ago when Dr. Wilmer brought up the subject, that I have not seen sudden extension of the blind portion of the field involve the central fixation point after trephining.

Long ago, I abandoned iridectomy in the trephine operation, contenting myself with a radial slit in the iris made by a snip with the scissors when the button of iris presents in the trephine hole. I still have under observation a patient on whom I operated in this way ten years ago. The slit in the iris is barely perceptible, but the tension remains well reduced.

That we should, and could with safety, instill atropin solution immediately upon finishing the operation, is one of the valuable contributions made by Elliot in reviving and reconstituting the operation of trephining All the old iridectomies for glaucoma which I have been able to inspect show posterior synechiae. Up to a short time ago, I agreed with those who thought the actual

cutting away of the iris in iridectomy contributed nothing to the tension reducing effect of the operation. I now believe that an opening in the sclera plus one thru the iris are requisites in all operations for the lasting reduction of intraocular tension.

The question whether we shall or shall not operate in a given case is, perhaps, more important now than the choice of a particular operation and is certainly even more in dispute. In the congestive type my voice would be for operation, with but few exceptions. In the acute attack it is imperative. In the varieties of this type we know that if the patient lives there is a certainty of blindness and suffering before him. Fortunately, there is no difficulty in obtaining consent to operate from the sufferer in the throes of an acute attack. We all know that this is not always the case with victims of the subacute or chronic variety, but knowing the future, I think we should exhaust all our powers of persuasion.

We today enjoy a great advantage thru the possession of the elegant and powerful alkaloidal miotics in being able to mitigate pain and other symptoms and to obtain favorable conditions for operation. Nevertheless, in my experience, no matter how carefully we use these remedies, notwithstanding gradual increase of their strength, the time comes when some contraction of the field of vision, some increase of tension, a little failure of sight, brings us face to face with an operation at last. Then the prognosis as to vision is poorer, the anterior chamber shallower, the iris atrophic and our difficulties increased. Individuals who die before this time comes are held up as examples of the superiority of miotic treatment, but of those finally driven to operation we hear less.

Unfortunately, all of progress is not improvement. More than once I have seen the relief given by miotics, even in an acute attack vigorously treated with an eserin solution, lead the patient to decline an operation; and, for the reasons given, greatly, I believe, to his disadvantage. No one doubts that the prodromic stage affords the best prognosis, yet how many promptly relieved by a miotic will then consent to surgical interference? It seems to me that we should keep constantly before our patients that miotics are but temporary measures mainly used to facilitate the coming operation. The exceptions to the rule that we should operate in congestive glaucoma will be dictated by common sense and the consideration of all circumstances. We might, for instance, spare a frail old person with but brief expectation of life from a dreaded ordeal by the use of miotics for a year or two. All this was the conclusion drawn by de Wecker from his great experience; my most modest confirmation may, perhaps, be of use.

If, to quote Graefe, once more (l. c. p. 337): "The prognosis is completely changed as soon as the field of vision is contracted," is true of the congestive, it is triply true of the simple type. In glaucoma simplex the state of the field of vision alone gives a picture of the condition of the eye, and is our one true guide to the progress of the disease. The central vision may be good and the increase in tension trifling but when an examination shows that the hitherto unaffected field is beginning to shrink, the time for operation is at hand. Tables giving the results of operations for glaucoma, especially simple glaucoma, which give no statement as to the condition of the F. of V., convey little information.

If now we are permitted to operate we can do the greatest good, for I believe that an *early* operation is as beneficial in one type of the malady as in the other, and that the disrepute into which operative interference in the simple type has largely fallen is, in the main, due to failure to give proper weight to the state of the field. We know that in the congestive type the changes in the optic nerve set up by the glaucomatous process, if once begun, are seldom if ever completely checked by the relief of tension. Cupping

of the disc appears early in glaucoma simplex and runs a long, slow course; so when injury to the nerve has already led to marked changes in the field when we first see the case, we have little to base a hope of delaying the progress of destruction upon. Only twice have I seen seemingly curative operations, both iridectomies, both in youngish negro women, made before any shrinkage of the field. In one under observation about a year there was no further progress of the disease; the other has retained vision of 20/XL for 17 years, and when heard from lately was carrying on her life in every way as usual. But her field is not greater than 20° in all directions.

In my experience it is nearly impossible to persuade the patient who consults us about an eye with a field greatly damaged by simple glaucoma to permit an operation on its fellow, with its field and vision still practically unimpaired. Yet, until I am convinced to the contrary, I shall believe that is the proper plan to pursue and that we might hope thus to delay indefinitely the blindness which, when we see these patients in their late thirties or early forties, is otherwise commonly inevitable. It is needless to say that exception to this, as to all such rules, may be dictated by many circumstances not always to be foreseen.

The question naturally arises as to how much loss of the field of vision should be regarded as precluding a resort to operation. Unhappily lack of experience makes a reply impossible, but for the present I should think that a loss equivalent to one third of the field would be very discouraging, and I should not operate save as a last resort in the presence of a steadily rising tension and shrinking field, in spite of the use of miotics.

OCULAR PHENOMENA IN ACROMEGALY.

WALTER I. LILLIE, M. D.

ROCHESTER, MINN.

The ocular findings and their relation to the sella turcica in fifty cases of acromegaly are here reported in tabular form. The principal points observed are also discussed, with the cases arranged in four groups. The paper is based on observations made in the section on Ophthalmology of the Mayo Clinic, Rochester, Minn.

The pathologic and structural changes in acromegaly have been widely discussed, and many cases have been reported which show the characteristic ocular involvement. The relative frequency with which the ocular mechanism is interfered with, however, has not been considered. The fifty cases reported here have been divided into four groups, in order to compare the ocular changes with the X-ray findings of the sella turcica. Definite acromegalic structural change were present in all. The general and neurologic findings are omitted, but the diagnoses are given.

GROUP 1. Nine patients had definite structural signs of acromegaly. The average duration of acromegalic signs was five years and four months. The sella turcica and the optic tracts were not interfered with. The perimetric fields were normal; and the slight upper temporal quadrant contraction was readily explained by the structural changes around the orbit and by the prominent eyebrows (Table 1).

GROUP 2. Four patients had definite signs of acromegaly, the average duration of which was four years and three months. The X-ray revealed normal sellae turcicae; but ophthalmic examination made of three patients revealed some change in vision, unequal pupils in one, and the pupillary reflexes slow and toxic retinitis in one. Retinal arteriosclerosis and slight venous congestion of the retinal vessels were also noted. One patient had slight exophthalmos, and another slight concentric contraction of the field of vision in the right eye. On X-ray examination the sella turcica of one patient (Case A363469) was reported to be normal, but on necropsy it was found to be size 4. All ocular findings in this group could easily be found in any series of cases, regardless of evidence of acromegaly. So these findings cannot be considered important as either diagnostic or pathognomonic of ocular changes in acromegaly, or even of chiasmal involvement. The fields are concentrically contracted for form and color in the right eye. The slight contraction in the upper temporal quadrant of the left eye is readily explained by the structural changes around the orbit (Table 2).

GROUP 3. Sixteen patients had definite signs of acromegaly. The average duration of the signs was four years. X-ray examination of the sella turcicae revealed enlargement 1 to 4, with erosion of the posterior clinoid processes. All but one patient had practically normal vision. One (Case A215790) had an amblyopic right eye which had been present from early childhood. One patient had unequal pupils. The fundi were all normal as were the perimetric fields. This is probably the most important group of the series, as it shows that even if the sella turcica has been enlarged or eroded by pathologic changes of the pituitary gland, the chiasm has not been sufficiently disturbed to produce changes in the ocular mechanism. The perimetric fields were normal both for form and color, except for the slight contraction in the upper temporal quadrants which was undoubtedly due to the structural changes of the face (Table 3).

GROUP 4. Twenty-one patients had definite signs of acromegaly. The average duration of the signs was six years and three months. X-ray examination of the sella turcica revealed enlargement 3 to 4, with erosion of the posterior clinoids. The ocular changes were not unlike those of any chiasmal lesion without acromegaly. The ocular changes may be summarized as follows: fourteen patients had visual changes; three had pupillary changes

either in size or contour of one or both eyes, eight had pupillary reflex changes, four had diminution in the pupillary reflexes in one or both eyes; two had definite Argyll Robertson pupils, and two had absolutely stiff pupils. Oculomotor changes were less common. One patient had poor convergence; one had horizontal nystagmus with absence of convergence; two had a definite strabismus varying from 20 to 30 degrees, and one had slight proptosis.

Five patients had normal fundi; four (Cases A119053, A204035, A-211582 and A247571) had changes which could occur in any series of patients without acromegaly or involvement of the chiasm. Five patients had simple optic atrophy of both discs; three had pallor of both discs, without loss of substance; and two had simple optic atrophy of one disc, the other being normal in appearance. One patient had a definite choked disc of one to two diopters in each eye; one had a blurred disc, the other being pale without loss of substance. The perimetric field changes of seven patients revealed partial or complete bitemporal hemianopsia; five patients had one blind eye, the other eye showing temporal hemianopsia; three patients had either partial or complete homonymous hemianopsia; two patients had concentric contraction in one or both eyes; one patient had a contracted temporal field, and four patients had absolutely normal fields (Talble 4)*.

One patient (Case A2147) on the first two visits to the Clinic gave no evidence of acromegaly. On her return seven years later, there was a definite history of structural changes for three years. X-ray examination of the sella turcica, and examination of the fundus disclosed evidence of chiasmal involvement.

One patient (Case A119053) on first examination had had acromegalic changes for five years, and X-ray examination disclosed a normal sella turcica and normal ocular findings. On her return four years later, the sella turcica was enlarged 3, and the fundus of the left eye revealed slight choroidal atrophy. The fields were normal. One year later, the patient returned and the fundus revealed bilateral hypertension sclerosis. The perimetric fields were again normal. Even with definite changes in the condition of the sella turcica, the ocular changes did not indicate chiasmal involvement.

One patient only (Case A374997) of the thirty-eight, on whom a Wassermann test was performed, had positive reactions on the blood. The perimetric fields revealed concentric contraction for form and color, and following treatment for syphilis the fields returned practically to normal.

SUMMARY.

Thirteen (26 per cent) of the fifty patients had normal sellae turcicae and ocular findings, so far as chiasmal involvement was concerned.

Of twenty-one (42 per cent) of the patients, the X-ray disclosed enlargement of the sella turcica 1 to 4, with erosion of the posterior clinoids and no ocular changes characteristic of chiasmal involvement. The condition of the patients in Group 3 is the most significant and interesting, at least from the point of view of the ophthalmologist. Forty-two per cent of patients whose X-ray examinations disclosed evidence of enlargement or erosion of the sella turcica had no signs of chiasmal involvement of the optic nerve.

Sixteen (32 per cent) of the patients had definite ocular changes characteristic of chiasmal involvement, in which the sella turcica was enlarged 1 to 4, with erosion of the posterior clinoids.

Sixty-eight per cent of all patients with acromegaly in this series were normal from the ophthalmologic point of view, so far as any chiasmal changes of the optic nerve were concerned.

*Data on these fields were published in the Jour. Amer. Med. Assn., 1923, LXXXI, 1765-1768.

TABLE 1.

Acromegaly With Normal Ocular Findings and Normal Sella Turcica

Case, date.	Sex, Age.	General and neurologic diagnosis.	Duration of acromegalic signs, years.	Blood Wassermann.	Basal metabolic rate.	Roentgen ray of sella turcica.	Vision of right eye.	Vision of left eye.	Pupils.	Reflexes.	Fundi.	Perimetric fields.	Ocular movements.	Operation.	Remarks.
A35039	F 36	Umbilical fistula: acromegaly	10			Negative			Normal	Normal	Normal	Normal	Normal	6-5-11 removal of umbilical fistula.	Death 3-9-13
A57754	F 47	Cystic adenoma of left lobe of thyroid; acromegaly.	7			Negative			Normal	Normal	Normal	Normal	Normal	8-31-11 thyroidectomy	
A91741	M 44	Furunculus; paranoia; epilepsy; acromegaly.	2	Negative		Negative	6/7	6/7	Normal	Normal	Normal	Normal	Normal	12-18-20 cellulitis left side of neck drained.	
A206411	F 47	Acromegaly.	3	Negative		Negative	6/7	6/7	Normal	Normal	Normal	Normal	Normal		
A225888	F 29	Enlarged thyroid; acromegaly.	1	Negative	+5 +4	Negative	6/6	6/6	Normal	Normal	Normal	Normal	Normal		
A238339	F 59	Multiple adenomas of thyroid; acromegaly.	15		+47	Negative	6/6	6/6	Normal	Normal	Normal	Normal	Normal	8-5-19 thyroidectomy	
A285194	M 32	Hyperthyroidism; acromegaly.	6		−15 −15	Negative	6/4	6/12	Normal	Normal	Normal	Normal	Normal		
A321390	F 11	Mental deficiency; acromegaly; giantism.	1	Negative	−15	Negative	6/12	6/5	Normal	Normal	Normal	Normal	Normal		
A370633	F 36	Polyglandular syndrome; acromegaly.	3		+19 +14	Negative	6/6	6/6	Irregular	Normal	Normal	Normal	Normal		

TABLE 2.

Acromegaly With Abnormal Ocular Findings and Normal Sella Turcica

Case, date.	Sex, Age.	Neurologic diagnosis.	Duration of acromegalic signs, years.	Blood Wassermann.	Basal metabolic rate.	Roentgen ray of sella turcica.	Vision of right eye.	Vision of left eye.	Pupils.	Reflexes.	Fundi.	Perimetric fields.	Ocular movements.	Operation.	Remarks.
A211021	F 54	Diabetes mellitus; acromegaly.	?	Negative		Negative			Normal	Slow	Toxic retinitis both eyes.		Normal		
A240602	M 51	Cerebral sclerosis; acromegaly.	?	Negative	−11 −9	Negative	6/6	6/20	Left larger than right.	Normal	Arterio-sclerosis of retinal vessels.	Normal	Normal		
A274715	F 42	Multiple adenomas of thyroid; acromegaly.	4	Negative	+1	Negative	6/12	6/7	Normal	Slow	Slight venous congestion of retina.	Left normal; Right concentric contraction.	Normal	6-25-19 partial thyroidectomy	
A363469	M 44	Adenomas of thyroid; acromegaly.	13	Negative	+20 +1	Negative	c. f.	6/7	Normal	Normal	Normal	Normal	Mild exophthalmos.	7-20-21 partial thyroidectomy	Death 7-22-21 Pituitary tumor. Hypertrophy of thyroid with adenomas. Sella enlarged 4.

TABLE 3.

Acromegaly With Normal Ocular Findings and Enlarged Sella Turcica

Case, date.	Sex, Age.	General and neurologic diagnosis.	Duration of acromegalic signs, years.	Blood Wassermann.	Basal metabolic rate.	Roentgen ray of sella turcica.	Vision of right eye.	Vision of left eye.	Pupils.	Reflexes.	Fundi.	Perimetric fields.	Ocular movements.	Operation.	Remarks.
A7475 3-7-08 6-1-14	F 52	Fibroid of uterus. Gallstones; acromegaly.	3			Enlarged 3			Normal	Normal	Normal	Normal	Normal	3-28-08 abdominal hysterectomy 6-13-14 cholecystectomy	
A15364 9-28-08 11-2-11	M 29	Acromegaly. Acromegaly.	3			Enlarged 4 with erosion of posterior clinoids.	6/6	6/6	Normal	Normal	Normal	Normal	Normal	11-9-11 Decompression for pituitary tumor	Death March, 1919
A63377	M 42	Hemorrhoids; acromegaly.	4	Negative		Enlarged 4	6/6	6/6	Normal	Normal	Normal	Normal	Normal		
A94510	M 29	Acromegaly.	4			Enlarged 4			Normal	Normal	Normal	Normal	Normal	Tonsillectomy 2-22-16	Death 2-16-15
A152061	F 39	Toxic goiter; acromegaly.	7	Negative		Enlarged 4 with erosion of posterior clinoids.			Normal	Normal	Normal	Normal	Normal	Thyroidectomy	Death 4-8-16 pituitary tumor.
A199217	F 19	Colloid goiter; acromegaly.	3	Negative	−3	Enlarged 4			Normal	Normal	Normal	Normal	Normal		
A215790	F 40	Multiple adenomas of thyroid; acromegaly.	?			Enlarged 1	c. f.	6/7	Normal	Normal	Normal	Normal	Normal		
A235445	F 52	Adenomas of thyroid; nephritis with hypertension; acromegaly.	?	Negative	+39 +18 +26	Enlarged 3	6/7	6/7	Normal	Normal	Normal	Normal	Normal		
A296117	M 32	Increased sugar tolerance; acromegaly.	5	Negative	+7	Enlarged 2	6/8	6/8	Normal	Normal	Normal	Normal	Normal	11-24-19 external and internal hemorrhoids removed.	
A300192	M 36	Chronic ulcer lower lip; acromegaly.	5	Negative	+17	Enlarged 3 with erosion of posterior clinoids.	6/6	6/6	Normal	Normal	Normal	Normal	Normal	3-2-21 excision of epithelioma of lower lip.	
A306605	F 54	Acromegaly.	5	Negative	+11	Enlarged 4 with erosion of posterior clinoids.	6/7	6/7	Normal	Normal	Normal	Normal	Normal		
A307854	M 40	Multiple adenomas of thyroid; acromegaly.	?	Negative	+13	Enlarged 3	6/5	6/5	Normal	Normal	Normal	Normal	Normal		

A344770	F 35	Acromegaly.	6	Negative	+7 +4	Enlarged 4 with erosion of posterior clinoids.	6/6	6/6	Normal	Normal	Normal	Normal	Normal		
A345987 1-12-21	F 31	Adenomas of thyroid with hyperthyroidism; acromegaly.	6	Negative	+86 +19	Enlarged 3 with erosion of posterior clinoids.	6/12	6/12	Right larger than left.	Normal	Normal	Normal	Normal	2-1-21 thyroidectomy of right lobe	
6-17-21		Acromegaly; unilateral pigmentation.				Enlarged 4 with erosion of posterior clinoids.	6/9	6/9	Normal	Normal	Normal	Normal	Normal		
A367428	F 25	Acromegaly.	4	Negative	+16	Enlarged 4 with erosion of posterior clinoids.	6/7	6/7	Normal	Normal	Normal	Normal	Normal		
A367535	M 41	Adenoma of thyroid; acromegaly.	9	Negative	+20	Enlarged 3 with erosion of posterior clinoids.	6/7	6/6	Normal	Normal	Normal	Normal	Normal		

TABLE 4.

Acromegaly With Abnormal Ocular Findings and Enlarged Sella Turcica

Case, date.	Sex, Age.	General and neurologic diagnosis.	Duration of acromegalic signs, years.	Blood Wassermann.	Basal metabolic rate.	Roentgen ray of sella turcica.	Vision of right eye.	Vision of left eye.	Pupils.	Reflexes.	Fundi.	Perimetric fields.	Ocular movements.	Operation.	Remarks.
A2147 9-24-07	F 40	Acute infection of breasts.												10-1-07 draining of left breast	
5-8-08		Ovarian tumor.												5-10-08 ovarian tumor removed	
3-27-15		Acromegaly; hemorrhoids.	3			Enlarged 4	20/30	l. p.	Left larger than right.	Right normal; left slow.	Right normal; left simple optic atrophy.	Bitemporal hemianopsia.	Normal		
3-27-17		Acromegaly.		Negative		Enlarged 4	20/30	l. p.	Left larger than right.	Right normal; left slow.	Right normal; left simple optic atrophy.	Bitemporal hemianopsia.	Normal		
A98762	M 31	Acromegaly.	3			Enlarged 4 with erosion of posterior clinoids.	Nil	20/40	Normal	Right slow; left normal.	Right simple optic atrophy; left normal.	Right blind; left temporal hemianopsia.	Normal		
A119053 11-13-14	F 44	Adenomas of thyroid; acromegaly.	5			Normal	6/6	6/6	Normal	Normal	Normal	Normal	Normal	11-23-14 thyroidectomy	
9-16-18		Acromegaly.			+14	Enlarged 3	6/7	6/7	Normal	Normal	Choroidal atrophy left eye temporally	Normal	Normal		

TABLE 4—Continued.

Case, date.	Sex, Age.	General and neurologic diagnosis.	Duration of acromegalic signs.	Blood Wassermann.	Basal metabolic rate.	Roentgen ray of sella turcica.	Vision of right eye.	Vision of left eye.	Pupils.	Reflexes.	Fundi.	Perimetric fields.	Ocular movements.	Operation.	Remarks.
6–4–19		Acromegaly.			+9 +9	Enlarged 3	6/7	6/7	Normal	Normal	Hypertension sclerosis both eyes.	Normal	Normal		
A154591	M 45	Acromegaly.	2	Negative		Enlarged 4	20/70	Nil	Normal	Right normal; left stiff.	Simple optic atrophy both eyes.	Right temporal contracted, left blind.	Normal		
A253290	M 28	Pituitary tumor; acromegaly.	6	Negative	+19	Enlarged 3; Calcified pineal gland.	6/10	c. f.	Normal	Normal	Simple optic atrophy both eyes.	Bitemporal hemianopsia.	Normal	1-25-19 pituitary tumor removed	Death 2-10-19 sarcoma of pituitary
A298149	M 37	Pituitary tumor; acromegaly.	2	Negative	+12 +2	Enlarged 4 with erosion of posterior clinoids.	6/10	6/10	Irregular	Argyll Robertson	Pallor of both discs.	Left homonymous hemianopsia.	Normal		
A311383	M 49	Pituitary tumor; acromegaly.	4	Negative	−26	Enlarged 4	6/12	6/30	Normal	Slow	Right disc pale; left disc blurred.	Bitemporal hemianopsia.	Normal		Death 12-14-21 at home
A328357 8–7–20	F 33	Adenomas of thyroid; acromegaly.	5	Negative	+29	Enlarged 4	Nil	c. f.	Normal	Stiff	Simple optic atrophy both eyes.	Right blind; left temporal hemianopsia.	Divergent squint 30°	8-31-20 removal of pituitary tumor	
9–13–20							Nil	6/30	Normal	Stiff	Simple optic atrophy both eyes.	Right blind; left slight return of temporal field.	Divergent squint 30°		
A334946	M 28	Pituitary tumor; acromegaly.	28	Negative		Enlarged 4 with erosion of posterior clinoids.	l. p.	l. p.	Left larger than right.	Slow	Simple optic atrophy both eyes.	Unable to obtain a field.	Divergent strabismus; proptosis.		
A347073 1–27–21	F 31	Pituitary tumor; acromegaly.	7	Negative	+17	Enlarged 3	6/6	6/10	Normal	Normal	Normal	Bitemporal hemianopsia for colors.	Normal	2-8-21 pituitary tumor removed	
3–4–21						Enlarged 3	6/6	6/6	Normal	Normal	Normal	Normal	Normal		
A361945	M 36	Pituitary tumor; acromegaly.	17	Negative	+8	Enlarged 4 with erosion of posterior clinoids.	6/7	6/30	Normal	Normal	Normal	Partial right homonymous hemianopsia.	Normal		
A374192	M 28	Pain right side of face; pituitary tumor; acromegaly.	4	Negative	+5	Enlarged 4 with erosion of posterior clinoids.	6/10	6/6	Normal	Normal	Normal	Temporal contraction.	Normal	11-5-21 removal of pituitary tumor	Death 11-5-21 cancer of pituitary extension into right gasserian ganglion.

A374997	M 51	Acromegaly lues latens.	6	Total inhibition	+33 +21	Enlarged 3 with erosion of posterior clinoids.	6/6	6/6	Normal	Normal	Normal	Contracted fields.	Normal		
4-24-22							6/10	6/7	Normal	Normal	Normal	Normal	Normal		
A173672 9-20-16	F 51	Acromegaly.	6	Negative		Enlarged 4 with erosion of posterior clinoids.	6/60	6/15	Normal	Normal	High myopia both eyes.	Partial bitemporal hemianopsia.	Poor convergence.	10-6-16 tonsillectomy	
10-13-21		Acromegaly.			+4	Enlarged 4 with erosion of posterior clinoids.	6/60	6/15	Normal	Normal	High myopia both eyes.	Left homonomous hemianopsia.	Poor convergence.		
A178603	M 26	Hemorrhoids; secondary anemia; acromegaly.	4	Negative	−3	Enlarged 4	20/50	20/50	Normal	Normal	Pallor of both discs.	Bitemporal hemianopsia.	Normal		
A204035	F 44	Hemorroids; acromegaly.	10	Negative		Enlarged 4	6/6	6/6	Normal	Normal	Retinal arteriosclerosis both eyes.	Normal	Normal		
A207068	F 55	Ventral hernia; hypothyroidism; acromegaly.	2	Negative		Erosion of posterior clinoids.	6/20	6/20	Normal	Normal	Mild choked disc both eyes.	Normal	Normal		
A210605 10-12-17	F 38	Pituitary tumor; acromegaly.	5	Negative		Enlarged 3	6/7	c. f.	Normal	Normal	Normal	Right normal; left concentric contraction.	Normal	10-25-17 removal of pituitary tumor	
1-14-20		Multiple adenomas of thyroid.			+36	Enlarged 3	6/7	c. f.	Normal	Normal	Pallor of both discs.	Bitemporal hemianopsia.	Normal		Death 1-25-20 recurrence of pituitary tumor
A211582	F 53	Multiple toxic adenomas of thyroid; acromegaly.	6	Negative	+33	Enlarged 4	6/6	6/6	Normal	Normal	Diffuse choroiditis both eyes.	Normal	Normal	10-29-17 thyroidectomy	
A245366	M 36	Tumor or cyst of pituitary; acromegaly.	1	Negative		Enlarged 4; complete destruction.	l. p.	m. o.	Normal	Right Argyll Robertson; Left normal.	Simple optic atrophy both eyes.	Right blind; left upper nasal quadrant intact.	Horizontal nystagmus; no convergence.	9-21-18 removal of pituitary tumor	Death 9-23-18 pituitary tumor malignant
A247571	F 57	Adenomas of thyroid; acromegaly.	3		+29 +35	Enlarged 3	6/20	6/30	Normal	Normal	Retinal arteriosclerosis both eyes.	Normal	Normal	10-16-18 thyroidectomy	

TONOMETRIC STUDY OF NORMAL AND ABNORMAL RABBIT EYES.

MABEL GREGG BOYDEN,

MADISON, WISCONSIN.

A general survey of what has been learned regarding intraocular tension by the use of the tonometer is given. To supplement what has been learned of intraocular tension in human eyes observations have been made on rabbits' eyes, both normal and those that were in various respects defective. These seem to show the limits of intraocular pressure in normal rabbit eyes and some wide departures from the normal in the eyes of defective stock. The studies were made in the Zoological Laboratory of the University of Wisconsin.

In 1905 Schiötz described his tonometer for the more accurate measurement of intraocular tension. Before that time and even up to the present, this tension was determined by pressure on the eyeballs with the clinician's finger and was registered simply as normal or relatively so much plus or minus the normal. This method obviously brings in a large personal factor involving such things as the experience of the clinician and the delicacy of his touch.

Since the presentation of the original Schiötz tonometer there have developed at least two different tonometers—the McLean and the Brown—working on the same principle but differing in detail and each claimed by its originator to be better than the Schiötz. A fairly large amount of literature has grown up dealing with the measurement of intraocular tension. These papers are chiefly concerned with the principles of tonometry and descriptions of tonometers whereas those dealing with experimental and clinical data are few. The need of an instrument like the tonometer arose from the fact that there is a normal range of intraocular pressure and that a number of pathologic conditions of the eye are accompanied, whether or not they are caused by, fairly large deviations from that normal. Sometimes this pressure rises considerably before severe permanent injury to the eye occurs; and so any means of detecting this change is of value.

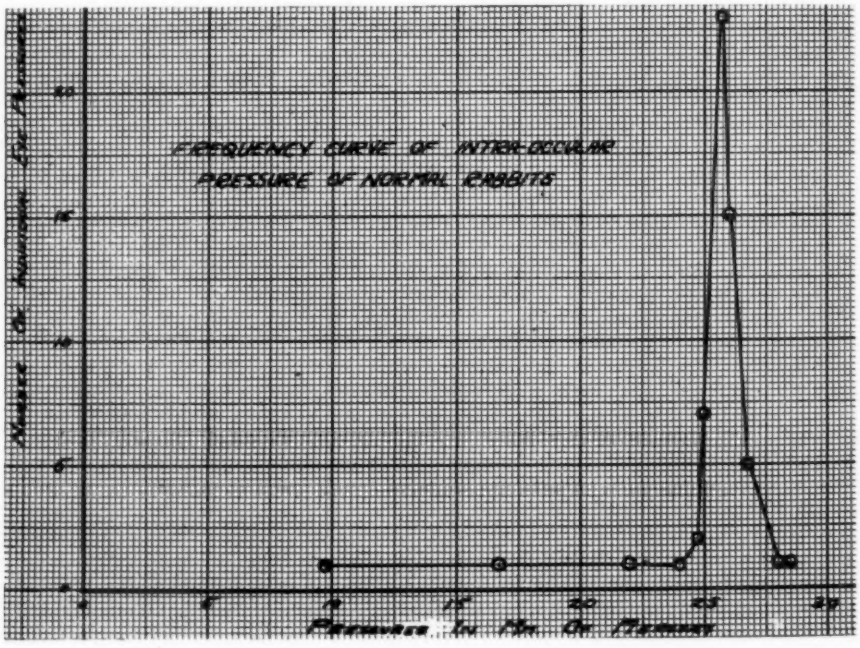


Fig. 1.—Intraocular pressure in normal rabbits most frequently a little more than 26 mm. of mercury.

The tonometer is an instrument designed to measure intraocular pressure. The eye is a spherical object and the foot of the tonometer is made concave of about the same curvature as the eyeball so as to fit smoothly over it. There is a hole in the center of the foot thru which a rod bearing a weight can freely pass. The rod is connected with a scale whose readings can be transposed into millimeters of mercury pressure. When the surface of the rod and the surface concavity of the foot of the tonometer are continuous, the scale reads at zero. If the eye is soft, the rod will dent into its surface and give a certain deflection on the scale. On the other hand, if the eye is hard it will not dent the surface so much and will give another deflection on the scale. The readings on the scale and the pressure transposed into millimeters of mercury are inversely proportional to each other. This is true because the harder the eye, i. e., the higher the internal pressure of the eye, the more resistance will be met and the more nearly it will bring the rod and the foot to making a continuous surface and so bring the reading nearer the zero mark on the scale. On the other hand, with a softer eye the converse is true.

In spite of the fact that nearly twenty years have elapsed since the description of the tonometer, not enough data have been collected to definitely settle the limits of the tension of the normal human eye. Schiötz sets the upper limit as twenty-five mm. of mercury as determined by his instrument. This figure is accepted as a conservative estimate by Priestley Smith, Weeks, and Brown. In many cases the tension can be lowered by the use of miotics.

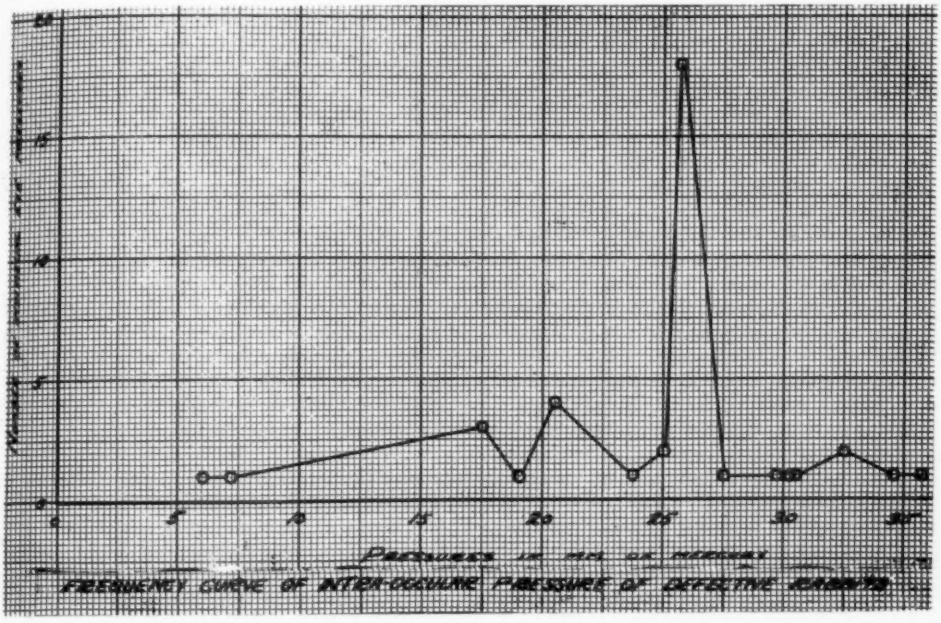


Fig. 2.—Intraocular pressure in defective rabbits. Maximum frequency the same as in the normal but many more deviations therefrom.

Gjessing reports in a rather comprehensive paper based on clinical findings of over a thousand eyes that the best weight to use on human eyes is the 5.5 gm., because larger weights tend to cause drainage of the humors and give inconstant results. He obtained an average of slightly under 20 mm. Hg. in 1,142 normal eyes and remarks that in young individuals with perfectly normal eyes the tension may register as low as 13 mm. or even 11.5 mm. Hg. He thus established a

lower limit for the normal range, in young eyes, than Schiötz set down.

Cridland reports only a very few cases of apparently normal eyes with a tension of 30 mm. Hg. with the 5.5 gm. weight but says that they later turned out to be glaucomatous. Gjessing reports only six normal eyes with a tension as high as 25 mm. Hg. with the 5.5 weight and considers them to be suggestive of the presence of glaucoma. He also considers, with Schiötz, tirely hold, because cases of glaucoma have been recorded which did not show any marked increase of tension. According to Dr. Davis this is probably transitory, the high tension again returning. A number of other conditions may, or may not, be accompanied by a rise of intraocular pressure. Among them may be mentioned uveitis or other uveal disease, tumor of the globe, and possibly also the bursting of the eye in advanced cases

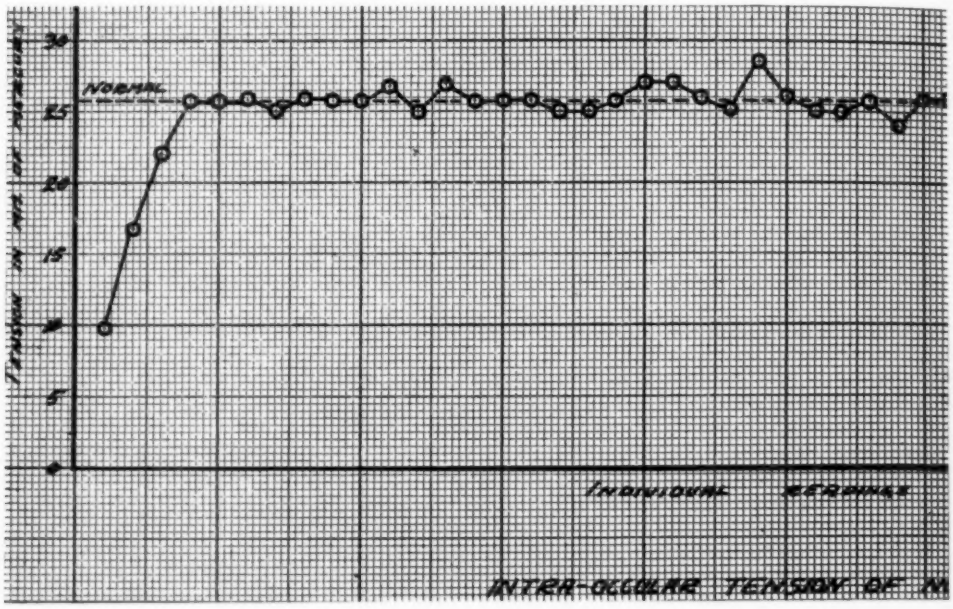


Fig. 3.—Intraocular pressures found

25 mm. as the upper limit. Two per cent cocain has no effect on the tension but five per cent tends to lower the tension. As has been pointed out before (Schiötz), comparison of tonometric and manometric reading do not absolutely agree; and so tonometer readings do not represent the pressure in actual mm. of mercury, but so long as tonometers are standardized this need be no drawback to their use.

Glaucoma is, perhaps, the most important of the diseases showing changes in tension. Formerly glaucoma was defined as a condition of the eye characterized by "increased intraocular tension with the causes and results of such increase." At the present time that definition does not en- of hydrophthalmia. In any event of marked increase of intraocular tension, for diagnostic purposes at least, the clinician may know that there is something wrong with the eye in question.

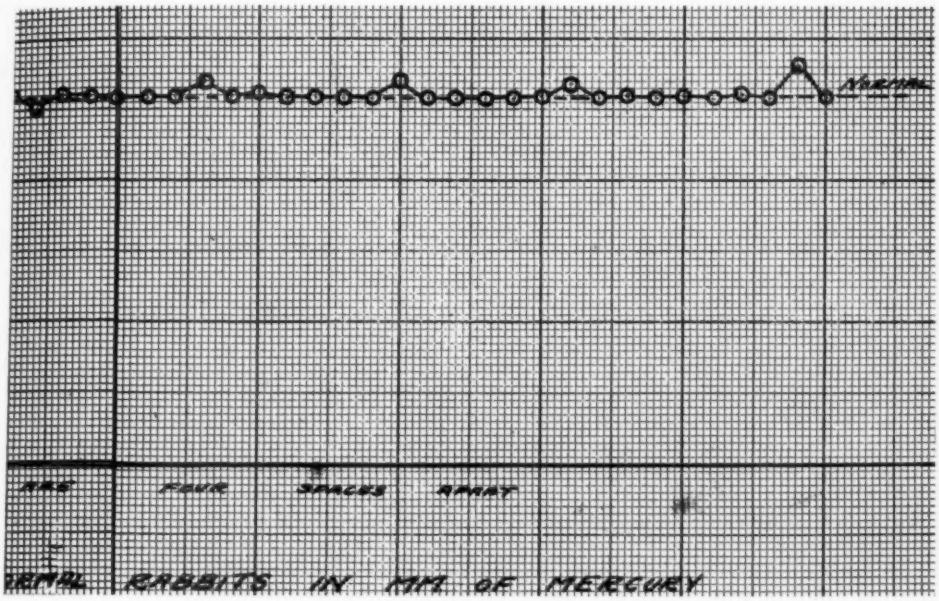
EXPERIMENTAL.

While working on some factors taking place in, or influencing the production of defective eyes in rabbits, such as those produced by Dr. Guyer and Miss Smith, at the suggestion of Dr. Lucien Howe, it was thought advisable to study eyes of defective strains and normal eyes with the tonometer.

The rabbits, with the exception of the first three or four, were all held by hand in such a position that when

the foot of the tonometer was placed directly over the center of the pupil the instrument stood vertical. The eyelids were kept open with the fingers, it being found easier than with a speculum. The eyes were anesthetized with four per cent cocain until the sensitivity was just lost, the measurements taken and the eye thoroly washed with boric acid solution. The whole procedure seemed to bother the rabbits not at all, as they were unusually quiet during the process. It was noticed that sometimes, immediately after the rabbit was in position for the measurement, the eye would bulge; but as the animal became accustomed to it the eye returned to normal. It was also noticed that some eyes took a relatively much larger amount of cocain than did others, before the sensitivity was lost—four or five drops. In all cases the figures here given represent averages, of five separate readings for each eye with the same weight. In many cases these were checked with readings with different weights.

In the table the rabbits designated as "normal stock" were all individuals in no way whatever related to the treated stock, or were pedigreed rabbits whose tensions were taken before any treatment was started. They were either bought from outside sources, or were bred from pedigreed stock of our own. Those designated as "defective stock" were individuals whose own eyes were visibly defective to the naked eye, or whose ancestors were defective eyed, or normal eyed stock of treated or defective lineage. All the individuals were full grown rabbits so



in individual normal rabbits.

the normal softness of young eyes was not a factor.

DISCUSSION AND CONCLUSIONS.

From the table it will be seen that the normal range of intraocular pressures is fairly constant, much more so apparently than that of normal human eyes. Of the sixty normal eyes recorded, forty-seven fall within the range of twenty-five to twenty-six millimeters pressure, six below 25 mm., and seven above 26 mm. In other words, seventy-eight and three tenths per cent fall in the first group; ten per cent in the second group; and eleven and sixty-six hundredths in the last group. Of the six falling in the second group, three were between twenty-four and twenty-

five millimeters of mercury. Of the seven falling in the third group, five fell between twenty-six and twenty-seven millimeters of mercury and none were above twenty-eight and one half millimeters of mercury. It seems reasonable, therefore, to conclude that the normal range of intraocular pressure should fall between twenty-four millimeters on the lower border and twenty-six or twenty-seven on the upper border. While the other eyes are apparently normal, incipient or real defects may be present and not distinguishable. This seems to be the case in the eye, the value of whose tension was nine and nine-tenths millimeters of mercury.

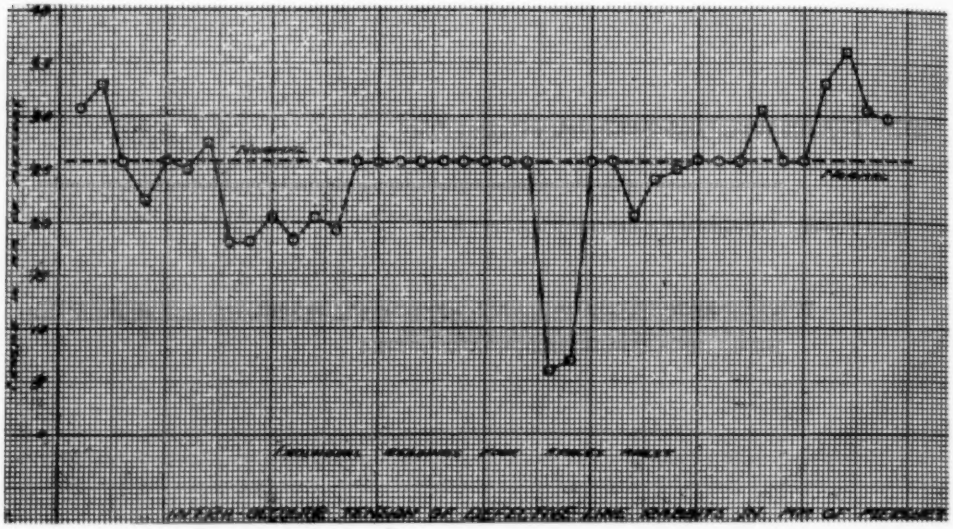


Fig. 4.—Individual readings in defective rabbits showing wide variations in tension.

In comparison, the measurements from the defective stock show some of them to be clearly within the normal range, but by no means all of them. There is much more variation which lies quite far above the normal more commonly than far below, altho some eyes were too soft to measure with any of the weights. Defective eyes may give normal tonometric values; and, vice versa, apparently normal eyes may give abnormal values.

From the data presented one can conclude the following:

1. The normal range for intraocular pressures of rabbits as measured by the Schiötz tonometer falls approximately between 24 and 26 or 27 millimeters of mercury.

2. The normal range of intraocular pressure of rabbits is apparently more constant and narrower than that of human eyes.

3. From the studies of intraocular tensions of defective eyed stock, or individuals of known defective eyed lines, it is not possible to point to a given tension as indicative of a given anomaly; but variations, especially the larger ones, may indicate abnormalities not detectable otherwise in the living state.

4. The use of the tonometer may lend itself to the detection of defective eyed individuals with apparently normal eyes in rabbits, as is possible in human beings.

It is with pleasure that I express my thanks to Dr. M. F. Guyer for opportunity to carry out these measurements, and to Dr. F. A. Davis and Dr. Lucien Howe of Buffalo for criticism and suggestions.

CHART OF TONOMETRIC MEASUREMENTS OF NORMAL STOCK.

Animal No.	Weight Used	Eye	Average Deflection	Deflection mm. Hg.
1	7.5	right	5.0	25.75
		left	5.0	25.75
N1	7.5	right	4.98	26.0
		left	5.1	25.0

P2	7.5	right	4.98	26.0
		left	5.0	25.75
B3	7.5	right	5.0	25.75
		left	4.8	26.75
P10	7.5	right	5.1	25.0
		left	7.9	16.75
B13	7.5	right	4.8	26.75
		left	5.0	25.75
B19	7.5	right	5.0	25.75
		left	5.0	25.75
P20	7.5	right	5.1	25.0
		left	6.1	22.0
P22	7.5	right	5.14	25.0 —
		left	5.06	25.75—
P26	7.5	right	4.9	26.5
		left	4.9	26.5
34	7.5	right	4.98	26.0
		left	5.1	25.0
P34	7.5	right	4.2	28.5
		left	4.92	26.0 +
P36	7.5	right	5.1	25.0
		left	5.2	24.95
P41	7.5	right	5.0	25.75
		left	5.4	24.0
P45	7.5	right	4.92	26.0 +
		left	4.98	26.0
P46	7.5	right	5.2	24.95
		left	11.1	9.95
P51	7.5	right	5.9	22.5 —
		left	4.98	26.0
P52	7.5	right	5.0	25.75
		left	5.0	25.75
P55	7.5	right	4.98	26.0
		left	4.86	26.75—
P56	7.5	right	4.98	26.0
		left	4.92	26.0 +
P57	7.5	right	5.0	25.75
		left	5.0	25.75
P58	7.5	right	5.0	25.75
		left	5.0	25.75
P59	7.5	right	4.86	26.75
		left	5.0	25.75
P60	7.5	right	5.0	25.75
		left	5.0	25.75
P61	7.5	right	5.0	25.75
		left	4.98	26.0
P62	7.5	right	4.78	26.75+
		left	5.0	25.75
N2A4	7.5	right	4.98	26.0
		left	5.0	25.75
N10A2	7.5	right	4.98	26.0
		left	5.06	25.75—
N10A3	7.5	right	4.98	26.0
		left	5.00	25.75
N11A2	7.5	right	4.5	28.0
		left	5.0	25.75

CHART OF TONOMETRIC MEASUREMENTS OF DEFECTIVE STOCK.

Animal No.	Weight Used	Eye	Average Deflection mm. Hg.		Cond't'n of Eye
N1B1	7.5	right	3.9	30.75	normal
		left	3.5	33.0	normal
N7B2	7.5	right	5.0	25.75	normal
		left	6.5	20.5	normal
N8B1	7.5	right	5.0	25.75	normal
		left	5.1	25.0	normal
N8B4	7.5	right	4.6	27.5	normal
		left	7.5	17.75	normal
N8B5	7.5	right	7.5	17.75	normal
		left	6.5	20.5	normal
Smith	7.5	right	7.5	17.75	normal
		left	6.5	20.5	normal
73A4	7.5	right	7.0	18.75	normal
		left	5.0	25.75	normal
74A2	7.5	right	5.0	25.75	normal
		left	5.0	25.75	normal
97B1	7.5	right	5.0	25.75	normal
		left	5.0	25.75	normal
97B3	7.5	right	5.0	25.75	normal
		left	5.0	25.75	normal
120A	7.5	right	5.0	25.75	normal
		left	5.0	25.75	coloboma of iris
121A2	7.5	right	14.0	6.0	microphthalmia
		left eye too bad to take.			
121C1	7.5	right	11.0	7.25	detached retina
		left	5.0	25.75	buphthalmia
121C2	7.5	right	5.0	25.75	normal
		left	6.5	20.5	normal
121C6	7.5	right	5.5	24 0	normal
		left	5.1	25 0	normal
123A1	7.5	right	5.0	25.75	normal
		left	5.0	25.75	normal
123A5	7.5	right	5.0	25.75	normal
		left	3.5	33.0	coloboma of iris
123A6	7.5	right	5.0	25.75	normal
		left	5.0	25.75	normal
121C5	7.5	right	3.2	34.75	normal
		left	3.0	35.75	normal
130A4	7.5	right	4.0	30 25	detached retina
		left	4.1	29.75+	detached

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THE VALUE OF FUSION TRAINING AND METHODS FOR ITS DEVELOPMENT.

DAVID W. WELLS, M. D., F. A. C. S.

BOSTON, MASS.

The importance of the fusion faculty to guide binocular movements and recognize the third dimension is pointed out. The value of such training in esotropia and heterophoria is emphasized. Methods of developing the fusion faculty by decentered lenses, stereoscopic pictures and bar reading are discussed. Results in 236 cases are reported. The dependence of success upon personal attention of the physician is emphasized.

Binocular single vision is a rather intricate psychic faculty, dependent on certain exact physical conditions. The first physical essential is the semidecussation of the fibers of the optic nerves, and the second is the proper coordination of the twelve extrinsic muscles, so that the retinal images shall fall upon corresponding points of the two retinas.

When it is realized that a deviation of less than a millimeter in the position of corresponding points means diplopia for small objects, it is almost inconceivable that any mechanics alone could secure the desired result.

THE FUSION FACULTY.

According to Worth, sight in the newborn is limited to fixation of a light, so that all the finer qualities are the result of personal experience. The involuntary movements of the eyes of infants would show that there is no conception of binocular vision. The full development of the fusion faculty is not attained by the normal child until the fifth or sixth year.

Physiologic diplopia means that objects nearer or farther than the point fixed are always seen double, but the adult has so far succeeded in ignoring this that it is sometimes difficult to make him realize the doubling. There can be little doubt that this fact plays a very important rôle in the child's experience; the fusing at different distances developing the idea of perspective.

The argument is here introduced to emphasize the fact that the third essential to binocular single vision is the psychic one of the fusion faculty. Without such an overruling guidance the necessarily exact coordination is inconceivable. The occasional clinical experience, that loss of sight of one eye is frequently followed by divergence, is an unanswerable argument for the importance of the fusion faculty in keeping eyes straight, and it plays quite as important a part in the production and the cure of those tendencies to turn, grouped under the name heterophoria.

THE THIRD DIMENSION.

A person devoid of a fusion faculty, or with only one eye, learns by experience to calculate distance by rapidly fixing the eye on objects to one side and thus establishes a parallax, which enables him to measure the distance by a sort of muscle sense. The inferiority of this sort of estimation of distances, compared with that possible to one with a well developed binocular fusion sense, was well shown by Howard at the Mineola experiment station. He claimed that many accidents in landing were due to the aviators' deficiency in estimation of depth difference.

Just how valuable this faculty is to chauffeurs, engineers and street car motormen has never been critically determined. When the facts are known is is quite possible that visual tests will not stop with measuring the acuity of each eye, and testing the color sense, but that a certain standard of stereopsis will be required. Four years ago the writer presented[1] a simple plan for the routine office measurement of stereopsis or depth difference, which requires only five minutes.

In 1890 Prof. Wells of Mass. Inst. of Technology found that some students had great difficulty in comprehending the diagrams in solid geometry and had stereoscopic charts made to overcome this deficiency. Several of these have been included in the author's selection of stereoscopic charts.

While a good fusion faculty is al-

ways a valuable possession, and in many walks of life absolutely essential, it is cheerfully admitted that one sees many patients totally lacking such a sense. These people use each eye separately and are not conscious of any loss, and have no asthenopia from incoordination.

Fusion faculty is also a matter of degree. Some patients are able to fuse concrete pictures by a rapid alternation of the two eyes, but have no conception of perspective. Other cases are able to fuse large concrete pictures and have some perspective but fail to fuse small types or characters. Simultaneous macular perception is essential to true binocular fusion.

The author is familiar with Gould's[2] theory of dextro- and sinistrocularity, but has not found it to coincide with the facts. Undoubtedly, many people who habitually suppress one eye may not suffer any annoyance from this condition. The same may be said of errors of refraction, but does any one for that reason consider astigmia physiologic? The writer maintains that the habitual use of the two eyes binocularly, with a minimum suppression of either, is as ideal as is the emmetropic eye to uniocular vision.

VALUE OF TRAINING FUSION FACULTY IN ABNORMAL CONDITIONS.

Esotropia: Every ophthalmologist is probably familiar with the pioneer work of Landolt, Javal and Worth. The question of whether the amblyopia causes the heterotropia or the lack of the fusion faculty causes the heterotropia with an amblyopia ex anopsia will always bring out a lively discussion.

Worth has certainly presented strong evidence that the vision can be maintained if the fusion be developed, but all who have followed his methods have found plenty of cases in which the amblyopic eye was not materially improved.

Landolt[3] sums up the matter as follows:

"Of course I have always considered the limitation of the temporal excursions of the eyes affected with convergent strabismus as the consequence and not the cause of strabismus. The abductors in this case lack exercise, especially in the symmetric movements. If, on the other hand, the absence of binocular vision is one of the principal causes of strabismus, the defective direction of the eye in turn becomes an obstacle to the development of this function. The individual thus finds himself in a vicious circle; he squints because his binocular vision is not developed, and binocular vision is not developed because he squints.

Tscherning[4] advises treatment under five headings:

"(a) Reestablishment of diplopia and if possible vision of strabismic eye.

"(b) Reestablishment of the approximately correct position of the eyes by way of operation.

"(c) Stereoscopic exercises.

"(d) Exercises without the stereoscope.

"(e) Controlled reading."

Certainly four of the above recommendations are fusion training.

In what proportion of cases esotropia can be cured by orthoptic means alone is difficult to answer, but it is certainly enough of a success so that in no case should one neglect to give it a good trial.

It is here that the amblyoscope of Worth is most useful, but great patience and intelligent parental cooperation are essential. Unfortunately one cannot watch the child's eyes and is entirely dependent on his correct report of what he sees. We still fail to get the cases early enough.

The value of postoperative training of eyes cosmetically straight has been advocated by Landolt, Tscherning and Javal for many years, and it is to be regretted that it is almost universally neglected. The remaining heterophoria may be more annoying to the patient than his previous heterotropia.

HETEROPHORIA.

Dr. E. Dyer, 1865, was one of the first to call attention to eyestrain from muscular imbalance. At the meeting of the American Ophthalmological Society[5] he read a paper entitled, "Asthenopia Not Connected with Hypermetropia."

Up to this time the teaching of Donders, that asthenopia (which we now call eyestrain) was always due to ciliary fatigue, was generally accepted. At the present time many of us believe that heterophoria is almost as frequently the cause as errors of refraction. When the two conditions exist it is often difficult to decide which is the more potent. The general rule seems to be to correct the refractive error, hoping thereby to relieve the heterophoria, but many times it is quite sufficient to correct the heterophoria.

This has usually been done by prescribing prisms either alone, or incorporated in the refraction correction, but, except for vertical errors, the writer usually reserves this as a last resort. Tenotomy and advancement are indicated in selected cases, but a discussion of surgical indications is beyond the scope of this paper. Exercises with loose prisms both at the office and at home have been very *generally advocated* and *occasionally practiced*, but have not been attended with any general or lasting success.

Heterophoria is the field in which fusion training is most important. The results in esophoria have not been very encouraging and less so in hyperphoria unless associated with exophoria. Exophoria, convergence insufficient and pseudoesophoria are most amenable. By pseudoesophoria is meant a condition in which the convergence duction is really weak, notwithstanding, all tests except duction show an esophoria. In the course of developing a better convergence a hyperphoria will often disappear, or such a tendency may be produced. Should it persist, it is corrected by means of a vertical prism combined with the lenses worn. It has puzzled the writer that Worth, who has made such extravagant claims for fusion training in curing heterotropia, should make so little use of it in treating heterophoria. Before treating the heterophoria, lenses are used for all they are worth, according to well established orthodox principles, unless the refractive error is slight and the heterophoria seems to be the active cause of the eyestrain. Javal, 1896, demonstrated the application of fusion training in heterophoria, which he calls latent strabismus.

METHODS FOR DEVELOPING FUSION FACULTY.

In discussing this subject in 1902, the writer made the following statement:

"The rapid development of the adduction which is often obtained by so-called gymnastics, strongly suggests that the gain is not a muscle hypertrophy but an increase in innervation, either in the responsiveness of the end-organ in the muscle or the convergence center or both. In the educational treatment of tabes, the incoordination is overcome by teaching the patient to gauge his motor impulses by the eye, in lieu of the normal sensory control. Repeated artificial contractions of the internal recti (the ciliaries remaining relaxed) establishes a habit of increased action, so they no longer lag when the impulse to converge and accommodate is felt. The coordinating center may also be taught to better appreciate the advantages of binocular perspective.

This is no special pleading but is analogous to other sensations. The pianist makes his fingers educate his brain, that the brain may do better work with the fingers. Tasks consciously performed are in time relegated to subconscious control. If this interpretation of muscle gymnastics be accepted, it is evident that the first indication is to teach the patient the fascination of true binocular fusion. All myology resolves itself into neurology and behind the nerve impulse is a fusion faculty."

Twenty years experience has not changed the writer's opinion of the main truth of this statement. Dr. Lancaster's very scholarly exposition of the "All or Nothing Theory"[6] is an important confirmation. He is undoubtedly right in arguing against a fusion center, and in demanding in its place the conception of a fusion faculty. His idea, that the development of this faculty does not mean increased strength of nerve impulse, but the utilization of a greater number of un-

used nerve fibers is a great help in understanding how it is done. But whether it be a stronger impulse, or a greater number of impulses, that is the usefulness on the presence of a fusion sense. But whether this sense is strong or feeble, real exercise is possible only by the patient making the effort."

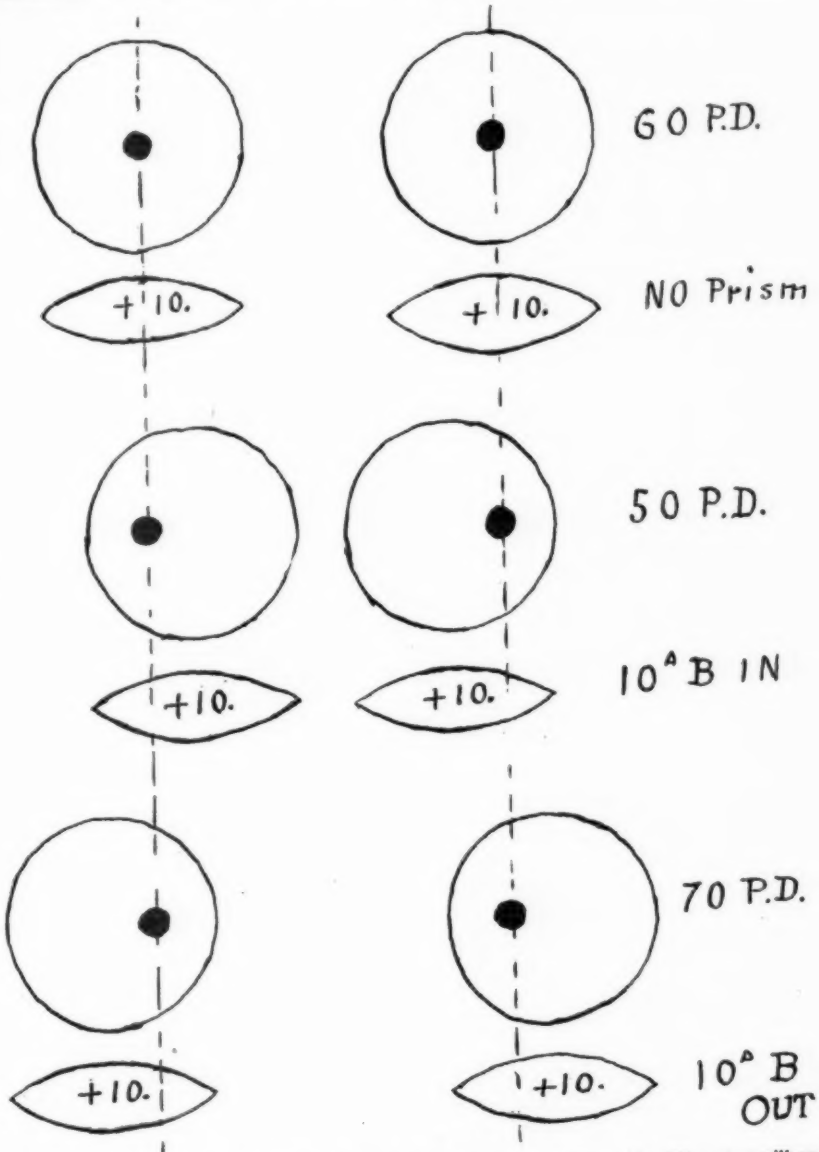

Fig. 1.—Diagrams showing prismatic effects obtained by decentering lenses with different pupillary distances.

essential for establishing well worn pathways of motor discharge, it is still more nerve impulse.

Dr. Jackson says,[7] "Orthoptic exercises are for nerve training—All forms of orthoptic apparatus depend for their

Since it is the consensus of opinion that more nerve impulse is the first essential, and that this is absolutely dependent on a fusion sense, and that the patient must *make the effort,* it seems evident that success depends

upon cultivating the fusion faculty by a method which incites the patient to make the effort.

For many years the writer has advocated stereoscopic training as the best means to this end. Many practitioners give to the patient a stereoscope and a set of cards and send him off "to work out his own salvation." This is a good deal like expecting a man to learn trigonometry by giving him a text book. The average man needs teaching, supplemented by the home use of the stereoscope, the cards selected so as to give him a definite lesson to learn. For office training the phorooptometer with two revolving prisms affords a simple means of making a stereoscope to order, to fit the patient's individual needs.

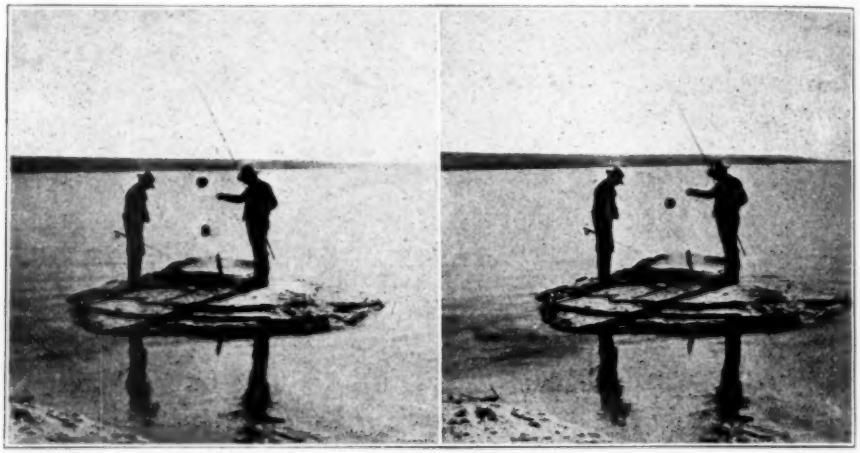

Fig. 2.—Stereoscopic pictures on which dots should be marked, two on the left and one on the right, to guard against suppression of one image.

Since, in treating convergence insufficiency, it is desired to associate a greater convergence impulse with the accommodative impulse, the tendency to accommodate is restrained by using +10. lenses and having the card holder fixed at 10 cm. distance. Any attempt to accommodate blurs the picture. The rotary prisms are adjusted so that the lines of sight cross at 6 cm. if one is using cards with corresponding points 6 cm. apart. Then concrete pictures are used commencing with those of large enough characters to be easily fused, and progressing to the more difficult. As soon as the concrete picture lacks any part of the two separate pictures, attention is called to the failure and the patient is helped to overcome this suppression by blurring the image of the dominant eye by an extra +lens inserted behind the +10. Or he is taught to partly close the dominant eye, so that the light to that eye is filtered thru the lashes. As soon as small characters are easily fused perspective cards are then added.

The cultivation of convergence requires *increasing the prismatic element of the stereoscope while the patient holds the image fused.* Naturally one would do this by turning the revolving prisms. Formerly the half pictures were inserted in the clips which by a right and left screw could be brought nearer together. Both of these methods were found to disturb fusion before much extra prism could be exhibited.

THE DECENTERING OF SPHERES.

The writer is indebted to Javal for the idea of insinuating the prism by decentering the spheres. With +10. lenses at 10 cm., each mm. of decentration is equivalent to 1^{Δ}, and with the regular size trial lenses 10^{Δ} to 15^{Δ} can be developed by increasing the pupilary distance of the phorooptometer. (Fig. 1.)

The patient will hold the image fused, by this method of introducing

prism base out, so very much better than by turning the revolving prisms or approximating the half pictures, that it is the keynote of success.

The fixed amount of prism is increased by turning the revolving prism while the patient's eyes are closed and the +10. lenses decentered in as much as possible. Then the decentering out of the spheres is repeated. When the patient can overcome the 60$^{\Delta}$ of the revolving prisms, extra prisms from the trial case are inserted in the clips. In this way 90$^{\Delta}$ can be exhibited.

For home work, as the convergence increases, prisms of 5$^{\Delta}$ and 10$^{\Delta}$ are inserted in the clips of the stereoscope which the patient uses. The average patient becomes keenly interested and makes the effort both at his office lesson and at home. He can note his progress himself.

At each treatment the patient's prism duction is tested with loose prisms held before the eyes, but he is never given prisms to use at home. This practice was discontinued many years ago, as it was discovered that some allowed an eye to diverge, suppressed the image, and thus developed a bad habit. The patient's eyes are watched while he is fusing the distant light, in order to detect divergence without diplopia. Fifteen minutes is enough time for giving a lesson, and the home work required is limited to fifteen minutes two or three times a day. At the beginning the patient is seen three times a week, then twice a week, then once a week, the interval being lengthened as progress warrants, so that the twelve or fifteen treatments required will consume about two months.

Fig. 3

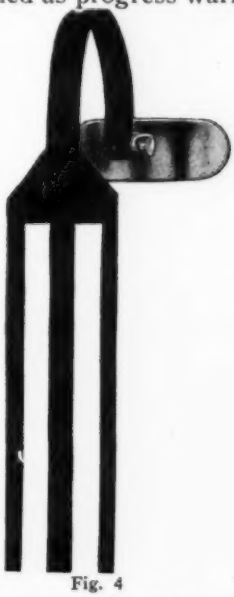

Fig. 4

Fig. 3.—Bar reading apparatus compelling use of both eyes in the reading of each line.
Fig. 4.—Similar apparatus with three bars compelling more frequent attention to images of each eye alternately.

MARKED PHOTOS OR STEREOGRAPHS.

To overcome the monotony and to keep up his interest, the patient procures a dozen or more good stereographs and these are marked with two dots on one side and one on the other, so placed that when properly fused, the three dots appear in a vertical line. This is to guard against a possible suppression of either eye. (Fig. 2.) A stereograph so marked is illustrated by Landolt in Norris & Oliver's System Vol. iv. p. 107. Such pictures for home use, with a pair of 10$^{\Delta}$ prisms, bases out, in the clips, cultivate fusion and convergence.

BAR READING.

While it is easy to demonstrate defect of fusion with the stereoscope, it

is impossible to know to what extent the patient is suppressing one eye in his ordinary reading. To guard against this, Javal constructed a grill of a number of bars, which rested on the reading matter on four legs. As this was a somewhat clumsy contrivance, the writer made a control device consisting of a strip of aluminum half an inch wide to be held about five or six inches in front of the face by means of an ordinary head band like those used for holding a mirror. This the patient uses for all reading, and with it in place he must, at least once in every line, use both eyes.

In many cases a triple control is used, but ordinarily the single strip suffices. The use of this is required for some months after the office visits have ceased. Tho annoyed by it at first, many patients come to recognize a distinct help in steadying the reading.

RESULTS.

In advocating a special method of treatment, it is realized that something must be said about results, but it is a somewhat difficult task to report accurately concerning therapeutic accomplishments. In 1917 the writer[8] published a schedule (Fig. 5) which it was hoped would be generally adopted, so that it might be possible to judge the relative value of different methods of treating heterophoria.

At this time 236 consecutive cases, which had been treated some years previously, were plotted on this schedule with the following result: (75% cured or relieved)

155 cases of exophoria at distance treated; 70 cases of exophoria or 41% symptoms cured; 82 cases of exophoria or 34% symptoms relieved but not cured; 14 cases of exophoria or 8% symptoms not relieved; 19 cases of exophoria, result not stated or treator treatment stopped—record incomplete. Assuming that these were not relieved would make the number of failures 33, or 21%.

81 cases of insufficiency convergence treated; 36 cases of insufficiency convergence or 44% symptoms cured; 34 cases of insufficiency convergence or 42% symptoms relieved; 7 cases of insufficiency convergence treated or 8% symptoms not relieved; 4 cases of insufficiency convergence, result not stated or treatment stopped—record incomplete. Assuming that these were not relieved would make 11 failures or 15%.

Case No.	SYMPTOMS H=Headache P=Pain in Eyes V=Vertigo C=Confusion	EXOPHORIA				DUCTION				Fusion Faculty	No. Treats.
		Phorom.		Cover.		Revolving Pr.		Loose Pr.	Stereo.		
		D	N	D	N	Ad.	Ab.	Ad.	"ONE"		

Fig. 5.—Heading of sheet for tabular

There have been some relapses (just how many it is impossible to say) but the great majority of the "cured" cases have retained sufficient power for all practical purposes. Patients are admonished to test themselves from time to time with the stereoscope and cards and if they notice any loss of power to renew the home exercises.

During the last twenty years the writer has had the pleasure of demonstrating this stereoscopic method to many ophthalmologists from various parts of the country, but very few have ever given it a serious trial. Many have been appalled by the amount of time required. Others have suggested

they would like to delegate it to their assistants, some have asked that their office nurses might be instructed in the technic. This attitude seems to minimize the importance of heterophoria as a cause of eyestrain. What is the trouble with the "rounder" who goes from one man to another to be given 0.25 difference in the strength of his lenses or 5° change of axis?

It is admitted that the nervous instability, (whatever that may mean) accounts for part of them, but unless the writer is blinded by his enthusiasm, heterophoria and defective fusion, or convergence insufficiency is the cause with the majority. If this be true (and their number is legion) are they not worth curing?

Dr Jackson says it is essential to "get the patient to make the effort," and who is best qualified to do this? Certainly not the assistant or the office nurse. The man whom the patient has been led to consult is the one who can do the most for him. The patient should receive his individual and undivided attention. There should be no third person in the room, and no distracting interruption. With the phorooptometer stereoscope one is able to watch the patient's eye while the lenses are being decentered, and if either eye is seen to fail to fix, the patient should be called up sharply with the words: "Careful, keep that left eye on the dot," etc. He will often express surprise that his lapse has been detected, and renew his effort to fix his attention. Besides he should be encouraged to try a little harder next time and commended when it is evident that the effort is being made, and he should have no doubt that *his success is a serious matter with the physician*. In short he should be given a hypnotic suggestion for all it is worth.

SYMPTOMS C=Cured >=relieved not >	PHORIA				DUCTION				% Loss after	REMARKS
	Phorom.		Cover.		Revolving Pr.		Loose Pr.	Stereo.		
	D	N	D	N	Ad.	Ab.	Ad.	"ONE"	6 mos.	

record of cases of heterophoria.

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DISCISSION OF THE LENS AFTER THE TWENTY-FIFTH YEAR.

RODERIC O'CONNOR, M. D.

OAKLAND, CALIFORNIA.

The general attitude on discission after the twenty-fifth year is stated, also the technic adopted. Curran iridotomy has been tried with discission and seems worthy of more extended trial. The disadvantage of discission is the duration of the treatment. Its advantages are safety, simplicity, better cosmetic results and absence of astigmatism following. Thirteen cases are reported in tabular form. Read before the Colorado Congress of Ophthalmology and Oto-Laryngology, August, 1924.

De Schweinitz, in his ninth edition, states that this operation is rarely done after the fifteenth year, Fuchs states that it is indicated only in children and adolescents, while other authorities place the upper limit at the twenty-fifth year. However, Beard has successfully operated, by this method, after the forty-fifth year and Herman Knapp at the thirty-seventh. Edward Jackson, at the A. M. A. meeting in 1916, reported in detail a number of cases so operated over the age of twenty-five and, in his conclusions, stated "For monolateral cataract up to middle life discission is to be considered as a proper procedure."

Since hearing Dr. Jackson's paper I have been encouraged to follow the conclusion, above quoted, with such good results that it has seemed worth while to place them on record with the hope that others may be encouraged to employ the operation.

In general I follow the technic as advised by Jackson and Beard, the important point being to make the needle puncture outside of clear cornea. In several recent cases I have combined, with the first needling, a Curran peripheral iridotomy in hopes that it would serve to lessen the pressure from undue swelling of the lens. It has seemed to work out that way; altho the absence of such symptoms, in the few cases so operated, may have been merely coincidental.

In using this modification butyn anesthesia should be used, or the pupil contracted with eserin, as it is practically impossible to perform the iridotomy with the pupil dilated. The iridotomy is done first, then the needle is turned and the lens punctured to its center. Even more liberty may be taken and the lens cut freely. This was done in case five with complete absorption after one needling and no washout required. In a number of older patients, where it has seemed worth while to secure a round pupil, I have done the Curran operation as a substitute both for a preliminary iridectomy and for the Chandler peripheral iridectomy. In those cases it was done as a preliminary operation. At the extraction there was no tendency of the lens to tear the small hole.

If further experience with the Curran iridotomy should verify my idea, it could be combined with the Homer Smith preparatory capsulotomy to prevent obliteration of the anterior chamber, which sometimes happens. It would add to the convenience of this procedure by permitting a longer interval to elapse between the capsulotomy and the extraction. Furthermore it might extend the usefulness of discission to include all immature cataracts. In these the soft cortex should absorb leaving only the nucleus to be removed, thru a relatively small and therefore safe incision.

The behavior of a case operated the day this is being written is the cause of the above suggestion. At the preliminary iridectomy the patient jerked at the moment of the incision, causing the point of the keratome to puncture the lens thru the iris. At the extraction, seven days later, the lens was so soft and gummy that it had to be practically scooped out. Irrigation was necessary to remove it completely. Even the nucleus was mushy altho the man was well past sixty. Had I known the condition, the lens could have been removed thru a small incision, as in the washouts after discission.

Several years ago Dr. Jackson, in an editorial stated that a method of breaking up the lens, so that it could be removed piecemeal thru a small incision, might finally prove to be the solution of the cataract operative problem. It

is barely possible that the above mentioned procedure might gain that end, at least in the early immature cases. At any rate I intend to try it out on the next suitable case. If successful the advantages of a simple extraction thru a small incision will be attained.

In order to save time the results in my thirteen cases will be given in a table appended hereto. It will be noted that the object of the operation, a clear pupil, was secured in all cases and that the only ones with reduced vision had a definite cause, outside of the operation, of which we were aware before operating.

I feel that this method should be thought of for all cases up to the forty-fifth year, possibly even later in immature cataracts. I have a woman under my care now who is blind in both eyes as a direct result of operations by the Smith-Indian method, when she was thirty-two years of age. There is little doubt that she would have good sight in both eyes had discissions been done or, for that matter, anything but an intracapsular operation.

CONCLUSIONS.

Disadvantages. The only specific disadvantage is the duration of the treatment. This could be materially shortened by stirring the lens freely and doing a washout in all cases.

The advantages are:

1. Safety.
2. Simplicity. It can be done as an office procedure from start to finish saving time, expense and absence from employment.
3. A better cosmetic result than any operation except a successful simple extraction.
4. A visual result possibly better than that of a simple extraction because of the absence of astigmatism.

TABLE OF CASES OF DISCISSION.

Number.	Age.	Type of case.	Needlings.	Washouts.	Duration treatment, months.	Visual results.	Comments.
1	28	High myopia	3	1	6	20/30	Pupillary region clear. Reduced vision due to myopic fundus changes.
2	30	High myopia	4	1	6	20/40	Pupillary region clear. Reduced vision due to myopic fundus changes.
3	30	High myopia	2	0	3	20/40	Pupillary region clear. Reduced vision due to myopic fundus changes.
4	27	Traumatic cataracts	3	1	3	20/20	Pupillary region clear in all three.
5	38	Traumatic cataracts	1	0	3	20/16	Pupillary region clear in all three.
6	48	Traumatic cataracts	0	1	3	20/20	Pupillary region clear in all three.
7	31	Congenital stellate	7	0	5	20/16	Pupillary regions clear.
8	31	Congenital stellate	2	1	3	20/16	Pupillary regions clear.
9	38	Congenital zonular	3	1	3	20/150	Pupillary regions clear. Double partial optic atrophy, noted before operation, was the cause of reduced vision.
10	38	Congenital zonular	3	1	2	20/40	Pupillary regions clear. Double partial optic atrophy, noted before operation, was the cause of reduced vision.
11	38	Premature senile cataracts.	5	0	7	20/16	Pupillary region clear.
12	47	Premature senile cataracts.	2	1	3	20/20	Pupillary region clear. Patient's misbehavior at washout prolonged it unduly, and resulted in moderate striped keratitis. Such behavior, in an operation, might have caused loss of the eye.
13	47	Premature senile cataracts.	6	0	12	12/70	Pupillary region and other media crystal clear. Macular degenerative changes, noted before operation, was cause of reduced vision. Developed, during an attack of flu, a severe iridocyclitis with hypopion, resulting in blockage of the pupil and finally requiring a scissors operation. Cause of prolonged duration of treatment.

NOTES, CASES, INSTRUMENTS

IRITIS WITH UNUSUAL FOCUS OF INFECTION.

WM. EVANS BRUNER, A.M., M.D.

CLEVELAND, OHIO.

The following case of iritis is of special interest only because of the unusual focus of infection.

Mr. C., age 57, consulted me November 23, 1921, with the history that the right eye had been inflamed a week. Three days ago he consulted an oculist in Kansas City who put some drops in the eye and told him to continue boric acid solution. Two days later while in St. Louis, he saw Dr. Wiener who found that he had an iritis with adhesions, but he had no time to study the etiology as the patient was leaving the city in a few hours for home. Dr. Wiener accordingly advised him to consult me at once upon his arrival in Cleveland. I saw him the following morning.

Examination showed considerable bulbar congestion, cornea slightly hazy with some deposits in the lower portion, iris muddy, pupil semidilated but not perfectly round, a complete circle of brown pigment spots on the anterior capsule of the lens, where posterior synechiae had evidently started to form—in other words, a typical case of iritis. Ophthalmoscopic examination showed the fundus hyperemic and hazy and compression of the veins by the overlying arteries.

Tonsils were not enlarged. The teeth looked fair and were later shown by X-ray to be in good condition. He was troubled with chronic constipation, but controlled this by salts every morning. He considered himself in good condition except for one thing—he had had a discharge from the bone of the right thigh for years. He had had a fracture of this bone many years ago, as a result of which a sinus developed. He had been operated upon first by the late Dr. D. P. Allen of Cleveland, and later twenty years ago, by the late Dr. Nicholas Senn. Each operation helped for a time, then the wound broke down and the discharge continued. For years he has noticed that if the wound becomes clogged so it cannot discharge freely, he feels badly and has a slight fever. Examination showed a very large scar to the outer side of the lower end of the thigh with some discharge.

I suspected at once that absorption from this osteomyelitis might account for the iritis and so told him. But he had had such a long period in the hospital upon the occasion of each former operation without permanent cure, that he was much averse to another operation. To be certain that there might not be some other focus of infection which would account for the inflammation of the eye, he was sent to the Cleveland Clinic for a complete general examination. They were "unable to find any cause for the iritis unless it could be the old infection of the femur." He was very desirous because of business reasons to postpone the operation for several months. Local and general medication were instituted tho I urged that operation upon the leg offered the best hope of clearing up the eye. As the inflammation had not improved by the twenty-ninth, the congestion being no less and the cornea even more hazy, he consented to operation. I saw him the following morning at the hospital before the operation, and the eye showed no change. Atropin of course was continued but all other medication stopped. Operation was performed by Dr. Crile. An incision was made along the old scar and dissection carried down to the femur. The medullary cavity was opened and considerable bone removed, though no sequestra were found. The bone cavity was packed with iodoform gauze.

The following morning the eye was looking better. On the second morning the redness was much less and the pupil larger, and on the third morning the redness was almost entirely gone with pupil dilated ad maximum the cornea clear, and the eye perfectly comfortable. Recovery was so prompt and complete, following the operation, that it seemed there could be no reasonable doubt that absorption from the diseased femur had caused the iritis. His recovery was uninterrupted except for one little flare up, due to absorption from a pocket of pus, which

was not properly draining; and he left the hospital January 11th.

March 30th refraction was tested with the following result:

R.+ 0.50 ⌒ + 0.25 Cy. 90° V = 6/5
L.+ 0.25 ⌒ + 0.25 Cy. 120° V = 6/5

He was ordered this, with +2.25 bifocals for reading.

Since that time he has had no further trouble with the eye.

A RANGE FOR REFRACTION EMBODYING NEW FEATURES.

SIDNEY L. OLSHO, M.D.
PHILADELPHIA, PA.

Sidelights, shadows, reflections, pictures, articles of furniture and irrelevant objects in general, all tend to divert the patient's attention during a refraction, and therefore prolong it, hasten retinal fatigue and lessen the accuracy of the result.

Figure 1 shows a refraction range in which these objectionable features, present in so many refracting rooms, have been overcome.

To provide this particular range, the wall toward a distally adjoining room was opened from floor to ceiling for a width of five feet. The light from two windows was successfully obscured without seriously affecting the ventilation. These windows were framed and curtained in a special prac-

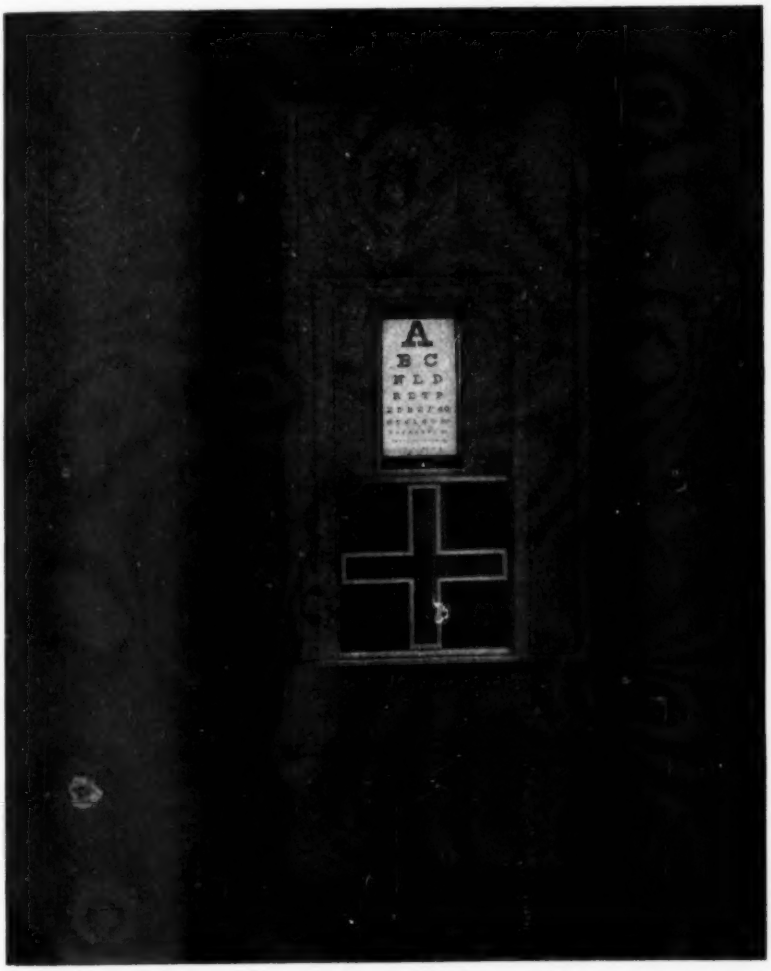

Fig. 1.—A Refraction Range showing a continuous unbroken, neutral-gray vista, leading to test chart cabinet and muscle lights.

tical manner, worthy of another illustration, see Figure 3. A side wall of sheet rock was constructed with a flush-fitting door near the distal end and a curtained doorway for the entrance of patients, near the proximal end. The room measures 5x24 feet.

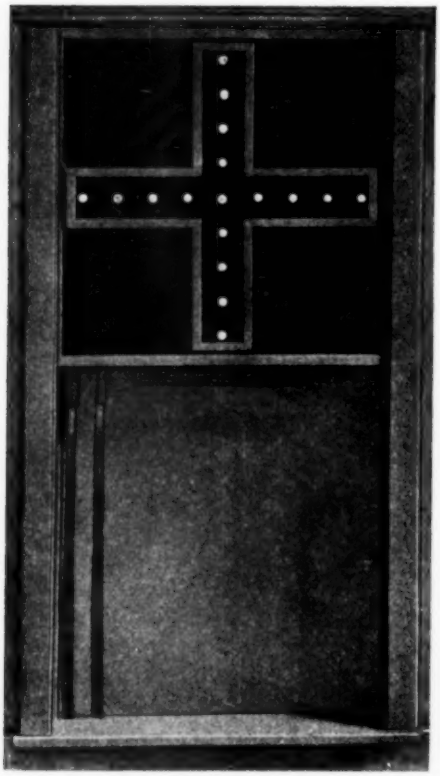

Fig. 2.—The Prentice Muscle Device set in a weighted sash, automatically illuminated when raised to the level of the patient's eyes.

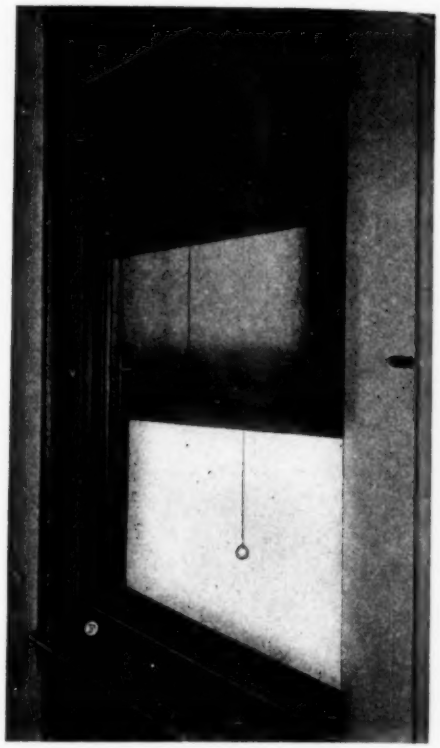

Fig. 3.—Method for obscuring all the light from a window by shades, yet permitting ventilation.

The test chart cabinet, the ceiling and walls are all a neutral flat gray. The linoleum floor is painted with a gray floor paint of the same shade. Test case stand and all the indispensable furniture are enameled gray. The object is to provide a continuous, unbroken, neutral-gray vista, leading to the objective, viz:—the Test Chart Cabinet and Muscle Lights.

The patient is seated in an operating chair, elevated sufficiently in this instance so that the examiner may stand to refract. The Test Chart Cabinet has been correspondingly elevated.

The cabinet for the test charts made for me by Messrs. Wall and Ochs, embodies a number of important new features referable to the illumination, the muscle tests and a screening device. The last can be better understood by reference to an article to appear shortly, describing it.

The charts are set far back in the cabinet and are illuminated by two pairs of 50 Watt "Mazda C" Daylight Lamps," controlled by a rheostat. Each pair is in a vertically placed W. & O. metallic reflector, covered by ground glass to diffuse the light. The reflectors extend the length of the test chart, one on each side, and are concealed from view in the cabinet. Each reflector is attached to a fixed vertical supporting rod, which acts also as a center of rotation so that the reflector may be made to face the chart at any desired angle. The reflectors are placed well to one side much farther from the charts than has been customary heretofore. The object is to illuminate the chart from an angle

sufficiently oblique that the great volume of incident rays of light will shunt off laterally inside the cabinet rather than be reflected at an angle of 90 degrees directly toward the eyes of the patient.

There is a sliding panel on the cabinet which may be lowered directly in front of the test charts. This panel has a small, adjustable, circular perforation which as the panel descends, is automatically illuminated by an independent lamp. This brings a single muscle test light on a level with the patient's eyes.

Figure 2. "Closeup" of Figure 1 shows the Prentice Muscle Device set in a weighted sash, raised to the level of the patient's eyes, whereupon it becomes illuminated.

Figure 3 shows a method for this or any other purpose, of effectively obscuring all the light from a window by shades, yet permitting ventilation.

"F" is a wood frame, 6 inches wide, ½ inch in thickness, resting on the sill, exactly fitting the inner window opening, secured in place by four small slide-bolts and easily removable.

The window shade already present is not disturbed. An additional shade as wide as possible is attached to the accessory wood frame backed up close against it. With both shades drawn no light leaks thru.

SYPHILOMA COMPLICATING AFTER TREATMENT OF CATARACT EXTRACTION.

E. E. Blaauw, Dr.

BUFFALO, N. Y.

This summer a well preserved man, 65 years old, came to Dr. Howe's clinic at the Buffalo Eye Infirmary. The right eye had been operated on about 4 years ago, and apparently an infection had destroyed whatever there was of vision. The left eye was normal except for a ripe cataract. Projection and light perception normal. Pupillary reaction good. As there seemed to be no contraindication the safest plan for the remaining eye was followed. A preliminary iridectomy was done and the eye healed well.

About six weeks later the patient was taken to the hospital and the lens extracted after opening the capsule. The nucleus was large and very little cortex remained behind. No vitreous escaped. The eye reacted very well and the patient was allowed to go home six days after the operation. He came to the clinic two days later and all seemed well.

Three days later, however, 11 days after the extraction, the iris showed a greenish hue in the lower half. Perhaps the injection around the incision was more than expected, where the eye did so well. Closer inspection showed in the bottom of the anterior chamber a lardaceous body, 3 to 4 mm. long; 1 to 2 mm. broad; within the middle a faint reddish tint.

As the patient was using mercurochrome the first thought was that this had penetrated into the anterior chamber. But the color was more suggestive of blood. It could not be hypopyon, as the surface was bosselated and not a straight line; and the general reaction of the eye was too mild for such a serious complication. Infection of the wound was absent, no infiltration. It could not be cortex, because no such large remnant had remained behind.

It could only be a new growth, and the diagnosis of syphiloma was made. The iris responded with an even dilation to atropin. With Gullstrand's slit lamp, the aqueous was found filled with tiny reflecting bodies in motion; many small round precipitates on the remaining lens capsule, and Descemet's membrane showed also very fine deposits. A blood specimen was taken for Wassermann and the patient received a salicydol injection. The next day K. I. was added to the therapy. From day to day the tumor could be followed growing smaller. Four days after the mercury injection the bloody streak had disappeared, the tumor remains could just be seen behind the arcus senilis, the shining particles in the anterior chamber were no more observed. The syphiloma had disappeared ten days after the first and 2 days after the second injection. The blood Wassermann reaction was negative.

From this clinical description it must be concluded that we were deal-

ing with a benign form of syphilis. How long ago the infection took place could not be determined, on account of the patient's mentality. Such a syphiloma has probably microscopically the appearance of granulation tissue. It seemed independent of the ciliary body, lying on the iris and in the anterior chamber. Its place at the limbus on the ciliary part of the iris was not specific.

A similar condition was seen last year at the clinic, situated on the iris sphincter, between 8 and 10 o'clock. The iris showed a well developed "collarette" "Krause" so that the sphincter part showed as a sort of gutter between this collarette and the pupillary pigment border. The patient, a woman 45 years old, showed a very painful inflamed eye. Specific treatment brought it to complete absorption, so that on discharge, after some weeks, nothing showed in the place of the large single syphiloma.

Our case is interesting on account of the time of development of the pseudotumor. The eye had reacted beautifully to two operations; still it can hardly be doubted that these traumatisms had provocative actions.

CONCLUSIONS. The slit lamp was of undoubted value in showing an inflammatory process of the interior part of the eye.

In this particular instance the syphiloma appeared diametrically opposite the place which was twice traumatised, and which showed no abnormality in healing. The other eye remained permanently quiet.

If, during the after treatment of cataract extraction, an iritis appears it is not necessary always to accuse infection, or remaining cortex of capsule. Years ago, before the days of the Wassermann reaction, in such a condition specific treatment produced the desired effect. The literature does not contain many references to this complication. I did not find any in the 1923 edition of Greafe-Saemisch, Augenärtzliche Operationslehre, nor in Igersheimer's book on Syphilis and the Eye.

THE PRISM SCOTOMETER.

JOHN N. EVANS, M.D.

BROOKLYN, N. Y.

When in the year 1668 the French priest Edmond Mariotte demonstrated the existence of the physiologic blind spot before the King of England, he had that monarch rotate his eye in various meridians until the original point of fixation disappeared and reappeared. Snellen and Landolt—in the first edition of Graefe-Saemisch—were still using his method. The method which has stood the test of time, however, has been that of maintaining the eye in steady fixation and moving the object, thus permitting accurate studies of form and relations.

A third method of outlining Mariotte's blind spot presents itself. If on a tangent surface we place a small white object 16½ degrees temporally from the point of fixation, we have located the center of the normal scotoma. If over the pupil of the fixing eye we now insert a prism in such a way that its edge approaches close to the line of fixation, we produce the optical effect of moving the center of the blind spot away from the object. It is obvious then that if a prism of about 8¼ degrees be used we would make the object appear at the edge of the blind spot and by turning the prism base in the opposite direction, we would make it appear at the opposite border. By using a prism of three degrees with its apex upward bisecting the pupil, we can define the upper border and using one of four and one-half degrees, apex down, we can locate the lower border.

Altho this is probably only a curiosity as a means of outlining the blind spot, we can see how it is possible to devise a small instrument of this nature to give readings accurately and rapidly. In a sense, it more nearly approaches objective studies than other methods, and one advantage lies in the fact that suggestion elements are reduced to a minimum as no moving object appears in the visual field.

(A rotating variable prism held in front of the fixing eye could be made to measure exactly any deviation required to bring the point of fixation to the edge of the blind spot. Ed.)

SOCIETY PROCEEDINGS

Reports for this department should be sent at the earliest date practicable to Dr. Harry S. Gradle, 22 E. Washington St., Chicago, Illinois. These reports should present briefly scientific papers and discussions, include date of the meeting and should be signed by the Reporter or Secretary. Complete papers should not be included in such reports; but should be promptly sent to the Editor as read before the Society.

SECTION ON OPHTHALMOLOGY, BALTIMORE MEDICAL SOCIETY.

October 23, 1924.

Symposium on Diabetic Retinitis.

Dr. Jessie W. Downey, discussing the subject from the viewpoint of the ophthalmologist, stated that most text books dealt with diabetic retinitis in a very dogmatic way. The matter is by no means so clear as their statements would indicate. Recent studies, especially by Wagener and Wilder, and by Benedict, had brought up the question as to whether diabetic as distinct from arteriosclerotic retinitis actually exists. Juvenile cases of diabetes, even in the most severe instances fail to develop retinitis. The metabolic disturbance alone does not seem responsible for the picture. On the other hand, typical retinitis punctata centralis is but occasionally seen in cases of arteriosclerosis showing no signs of diabetes. These cases, however, are extremely rare as compared with true diabetic retinitis, and if it is to be assumed that the arteriosclerosis alone is responsible for the retinitis in diabetics, it is difficult to understand the rarity of the picture in a condition as frequent as arteriosclerosis without diabetes. It would seem that both factors must be operative in the usual case of diabetic retinitis. The whole subject is much complicated by the fact that neither diabetes nor diabetic retinitis are single disease entities, tho our knowledge of the distinction between different subgroups is as yet imperfect. Some light may be had from a careful correlation of the ocular and general condition and from pathologic studies.

Dr. Eugene Leopold, speaking from the viewpoint of the internist, said that the arteriosclerosis of diabetics was of the usual senile type and that the retinitis occurred only in elderly and arteriosclerotic cases. Neither hyperglycemia nor acetonemia could cause the retinal lesions. The diabetes of elderly persons has been ascribed to arteriosclerosis, but the case is not absolutely clear, for there are many arterioclerotics without diabetes. Obesity, he thought, is a very important factor in the etiology of diabetes. In diabetic retinitis he felt the arteriosclerosis was the important element, the diabetes secondary.

Diabetic retinitis is not an ominous symptom as regards the prognosis of diabetes. It can be taken as a valuable index of the general vascular condition, but the diabetes in these cases can usually be expected to be mild. Equally, the control of the diabetes can hardly be expected to have any direct influence on the course of the retinal disease. Nevertheless, cases with retinitis should be subjected to specially careful medical care, including the best antidiabetic treatment for the development of the arteriosclerosis may be somewhat retarded and while the retinal lesions rarely recede, their extension can sometimes be stopped.

Formerly, high protein diets were used in the treatment of diabetics. This was bad for the arteriosclerosis, for the kidneys and for the retinal lesions. In the modern Woodyatt diet, however, little protein is used and this is the best treatment for the retinitis as well as the diabetes. Insulin is rarely needed in these cases in which the diabetes is relatively mild.

Dr. Jonas S. Friedenwald discussed the pathology of diabetic retinitis. Surprisingly little is known about this condition. Pathologic studies have been made on very few cases, and these have shown little beyond the hemorrahages. Special study of the blood vessels has been reported in one case without any significant findings. The white spots are supposed to be similar to those seen in albuminuric retinitis, and are said to

contain cholesterol, but this too is unproven. Dr. Friedenwald had studied pathologically the eyes of two diabetics, but no lesions were found nor had there been any retinitis visible ophthalmoscopically in these cases.

Perhaps some light may be thrown on the relation between diabetes and arteriosclerosis in the causation of retinal lesions when we know more about arteriosclerosis. At present we diagnose arteriosclerosis without questioning whether the larger or the smaller vessels are involved; tho variations in the localization of the lesions may result in quite different clinical pictures. The part played by changes in the capillaries in the production of different forms of retinitis has as yet not been studied.

Discussion. Dr. C. A. Clapp said that the question of the etiology of diabetic retinitis had aroused attention as far back as the seventies. Nettleship and others of his generation engaged in a lively discussion as to whether diabetic retinitis existed as a separate entity distinct from nephritic retinitis.

The youngest case reported by Mr. Foster Moore was 39 years old. Dr. Clapp had, however, seen a typical diabetic retinitis in a patient aged 34. Since the publication of the work of Wagener and Wilder, he had studied with special interest a series of cases from the metabolic clinic at Hopkins. Typical retinitis was very rare, but punctate retinal hemorrhages, especially in the peripheral parts of the fundus, were relatively common. This was contrary to the general teaching that the seat of the lesion is usually central. The part that might be played by lesions of the capillaries was interesting. Zellers has maintained that the conjunctival capillaries are dilated in diabetes.

Dr. Harry Friedenwald said that in the last eight years he had seen in his private practice 16 cases of diabetic retinitis. The average age of these cases was 61 years. All cases but one were of the central punctate type; one was of the nephritic type. Marked disease of the retinal vessels was noted in many, high blood pressure in some, two cases were complicated by chronic glaucoma, and several by cataract. The diabetes alone cannot cause the retinal changes, as is shown by the absence of these changes in young diabetics. However, the diabetes must play a definite part in the production of these lesions, otherwise it is not possible to explain the relative rarity of this ophthalmoscopic picture in arteriosclerosis without diabetes.

Doctor Goldbach said "the lesions in diabetic and advanced arteriosclerotic retinitis are somewhat similar, and it is dfficult to say definitely whether the diabetic or the cardiovascular condition is the greater factor as the underlying cause of the retinitic changes."

Dr. Fleck told of a case in which there was much debate as to whether the retinal lesion was of the diabetic or nephritic type. Finally the urine was examined and both albumin and sugar found.

In concluding, Dr. Leopold mentioned the cases of transient amblyopia at the beginning of antidiabetic treatment. These had been attributed to acidosis, but such cases had come about even under the Woodyatt method of treatment, in which no acidosis was produced. Indeed, he had seen a case in a diabetic with marked acidosis who developed transient amblyopia when both his glycosuria and acetonuria were disappearing.

Jonas S. Friedenwald,
Secretary.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY.

October 21, 1924.

Dr. Clarence Rubendall, presiding.

The program was taken up largely with reports on the recent meeting of the American Academy, at Montreal. The ophthalmologic reports were made by Dr. Nora M. Fairchild and Dr. F. W. Dean; the oto-laryngologic reports by Drs. Uren, Haney, Wherry, and Rubendall.

Central Retinitis.

DR. F. W. DEAN reported a case of unusual central retinitis in a woman twenty-five years old. When seen in February, 1923, she had a small patch of choroiditis above the left macula. She showed considerable muscular imbalance which varied during the next two or three months in the successive examinations. On account of headaches, the removal of infected tonsils under local anesthesia was advised. Three months later a tonsillectomy was done by the family physician under general anesthetic. Six weeks after the operation, vision of the left eye was 20/200 and the macular region showed distinct congestion. Three days later there was a circumscribed edema at the macula with hemorrhagic spots around it. Vision was reduced to light perception. Four days later the typical appearance of albuminuric retinitis was present. Urinalysis now showed a trace of albumin, altho the patient stated that previous analysis had been negative. She was put on a nephritic diet. When last seen the macular region showed a peculiar cone shaped elevation resembling a figure in de Schweinitz' text, described as "central cone shaped lesion in the choroid surrounded by lines of edema." It was thought to be a peculiar manifestation of albuminuric exudate in the retina, probably brought on by the general anesthetic.

SANFORD R. GIFFORD,
Secretary.

NETHERLANDS OPHTHALMOLOGICAL SOCIETY.

Sixtieth Meeting.

DR. G. F. ROCHAT, presiding.

Translated by E. E. Blaauw, Buffalo.

Faulty Registering of Different Tonometers.

J. H. A. F. TRESLING said that on account of the difference in cost more German instruments are imported, especially tonometers. Such an instrument should always be compared with one having a register known to be accurate.

Exactness of Principle and Method of Examination of Colors.

P. J. WAARDENBURG demonstrated two instruments which he had constructed to make it possible for ophthalmologists to practically execute in a short time admixtures of colors, comparisons of colors reciprocally and with white light of different intensity, and to make determinations of the threshold values of sensitivity for certain colors.

One instrument should be called an anomaloscope. A wooden box lined with asbestos, 36 cm. by 15 cm., and 8 cm. was divided by placing in the center thereof (exactly fitting) a box of 12 cm. by 9 cm. and 7 cm. Across this box are two parallel round tubes of white celluloid or frosted glass. They pierce the opposed walls and can receive light from both sides, because of electric lights that are in both ends of the large box. The box is closed with a slide in which there are two lengthwise slits in the middle above the tube. The large box is covered with a plate, which closes the 3 subdivisions from each other, airtight.

The following experiments can be made with the instruments: In the first tube, spectral red light is admitted by closing one opening with a special red glass and the other with spectral green. These colors become less saturated toward the middle and a short neutral intermediary transition zone is present. This point, which is neutral with a blue greenish glass and more yellowish with a yellow green glass, is found by moving a small disc with a small central opening (2.5 or 4 mm.) horizontally over the slit. Anomalous individuals will locate other places than normal, too much toward the green side (deuteranomalous) or too much toward the red side (protanomalous).

After the determination, a comparison must be made with a point of the second tube, over which a similar small tube is moved. This tube is light tight at one end; but receives light at the other end, thru a small central opening in the disc, which closes the tube. The size of this opening must be determined experimentally. Sufficient light must be allowed to enter so that the decreasing light intensity in the tube is such, that the illumination at the side of the light source is clearer, and at the other side darker than that of the neutral point of the other tube. If the light is too yellow the tube can be covered by a blue glass plate. This is

especially necessary with celluloid tubes, which accentuate the yellow in the mixed light. In this way colors can be mixed and a "Raleigh comparison" made as well as a comparison of clearness (in using complementary colors for white, and more nearby colors for yellow), which will detect the anomalous trichromats.

Protanopes and deuteranopes are found by letting them make comparisons between red and green, red and grey, and green and grey. This is done easily with different discs that close the tube. In one tube red, and the other tube green light can be thrown and the opposed side closed to light. In dealing with a red or green blind person the rule found by v. Hess holds, namely: that an equality of red and green (green blind) is also valid for a red blind when the light intensity of the green sinks to about 2/5. If red and green light enters the two tubes from the same side the red blind will make an equation between two rather distant points; the green blind between two points which lie nearer each other.

The degree of color sensitiveness can be determined by throwing colored light from one side in the tubes and white light from the other side. By moving the diaphragm over the tubes the moment of first color impression can be determined. Oversensitiveness as well as undersensitiveness for one or more colors can be demonstrated. A carefully selected blue filter can serve to show the diminished sensitivity for blue by red-blindness, as was found by Hering and Hess. A classification of the anomalies can be made in the same way with red and green filters. It can be determined if anomalous persons are hypersensitive for one or more colors if their anomaly results from hyposensitiveness for one or more colors, or if the decrease keeps pace for different colors.

Waardenburg believed that, after slight improvement, with this instrument more could be attained than with the anomaloscope of Nagel. This determines only existing inequality of sensitivity for red and green but not of its quantity, nor of a hyposensitiveness for red or green, if present, and far less for blue. Both instruments vary two factors while determining the neutral mixture, that is, the one color decreases in the same measure as the other increases. For measuring a color defect it is desirable that only one factor change. We therefore replaced the tube box by a much smaller metal tube, with an opening upward of 6 mm. Within the metal tube, one of celluloid was placed and in this, on both sides of the upward opening of the metal tube, a green and a red glass were placed at a distance from each other of 1 cm. If the middle opening receives an equal amount of red and green then its color is neutral. The mixture is made by moving one of the lamps horizontally. The one which gives too much colored light is moved backward. Anomalous persons make other determinations than normals and, as only the distance of one of the light sources is changed, a measure of the deviation is found.

The second instrument, which Waardenburg presented, was a box 17 by 11 cm. and 4.5 cm. high. The cover contained two rows of five openings. A valve which moved around a lengthwise axis, divided the box into two equal halves, which were then separated and made light tight. Toward the rear in the right side of the box was a lengthwise slit, in which a 4 volt lamp could be moved and which was to appear successively behind each one of the 5 openings. At the same time a disc with two openings moved forward over the box each time, leaving free 2 openings next to each other opposed to the lamp. The right row of openings was closed by lateral, movable green celluloid discs, which were chosen so that the first one permitted spectral green; the second only extreme spectral red; the third red as well as complementary green, but predominating green; the fourth the same with predominating red; the fifth red and green in equal proportions up to neutral. The left row of openings was also illuminated on the right by a 4 volt lamp, its intensity regulated by turning of the valve. The yellow from the light was neutralized by gray walls and by a blue movable wedge in a lengthwise direction (according to Hess) before the opening. Before the right side openings, green and red wedges were movable. In

this way it is possible to make neutral comparisons and a distinct comparison with the next opening. The fifth right opening was seen by anomalous people as colored and could be made neutral by them with one of the wedges. By exchanging the blue wedge at the left side with the red or green one, a comparison between red and green could be made, and the degree of light intensity, judged by the amount of valve motion, made it possible to separate the protanopes from the deuteranopes. The threshold for colors can only be determined by a rheostat which can dim the light almost entirely and then slowly increase its intensity.

Both instruments must be used in the dark room; investigator and patient observe at the same time and in the same adaptive condition. Anomalies by a changed resorption in the lens or macula lutea can thus be detected, and at the same time the influence of centric and excentric vision is observable.

Waardenburg related the incident of the railroad disaster of which Hess was supraarbiter. The engineer had satisfied all examinations, but showed with delicate methods a subsensitiveness for colors.

Discussion. Weve preferred Hess and Waardenburg's methods, but defended the anomaloscope which must be considered the highest test: Many do not know how to use it.

Waardenburg believed that not all forms were detected hereby.

Weve replied that it is sufficient for practical purposes in that it discovers the color anomalous individuals.

Tresling observed that anomalous trichromates can escape with the anomaloscope, if they can observe long enough; but with very short observation they are detected on account of the abnormal contrast.

Waardenburg agreed with Hess that anomalous trichromates do not have an increased contrast.

Akinesis for Ocular Operations According to the Method of Van Lint-Rochat.

H. F. Dubois referred to the communication of Rochat in 1920 and of his use of the Van Lint-Villard method, advocating injection of novocain in the orbicularis muscle. Rochat had used the method more generally and added injection in the belly of the superior rectus muscle to prevent upward motion of the eye during operation.

Dubois has used the latter method in 29 cases: 18 lens extractions of all kinds, 2 linear extractions, 8 iridectomies and one case of reclination. The iridectomies performed were in acute inflammatory glaucoma and seclusion of the pupil. One linear extraction was in a child of 6 years. He considered it unnecessary for trephining and tenotomies. The only objection is the 20 minutes waiting before the areflexia is completed.

Discussion. Weve also used the method and is surprised that Dubois has to wait 20 minutes after the injection. He uses a 5% solution of novocain, which he considers innocuous for the tissues. Only once has he had vitreous prolapse. Altho the muscles were well paralyzed, he attributed this to large quantity injected in the superior rectus (1.5 cm. novocain), which could have increased the intraocular tension.

Marx injected for Barraquer's extraction, without injecting into the superior rectus, as the eye does not need to be turned downward.

Reiling also did not understand the long waiting. He found it was not necessary to inject in some cataract extractions, when good assistance was available.

Tresling mentioned Duverger's method and referred to a case of paralysis of the external rectus which resulted from the injection.

Van Kleef has obtained good results from 1% novocain.

Dubois used stronger concentration and waited a shorter time. He insisted on using the method in all extractions, as one never knows beforehand the condition of the zonule of Zinn. He considered Duverger's method dangerous; a case of subsequent neuroparalytic keratitis, with loss of the eye, had been reported in the literature.

Indication for Reclination of the Lens.

H. F. Dubois thinks the only indication for reclination is the danger of expulsive hemorrhage. All writers prefer reclination to depression. Dubois performed the operation in 2 cases: 1. A

63 year old woman with a long standing serpent ulcer of O. D. had a left ripe cataract, and a bilateral dacryocystitis with much secretion. The day after coming into the hospital both tear sacs were removed after which the ulcers healed quickly, but with leucoma adherens and only light perception. Twenty-one days later, preliminary iridectomy was done on the cataractous eye; slight subluxation of the lens downward became noticeable and the vitreous presented. As the zonule was defective upward, vitreous prolapse was certainly to be expected after the section. Ten days later reclination by scleronyxis was done. The patient went home ten days later with vision of 2/10 with + 11. D. After 3 years the eye was still quiet. Vision had remained the same and the lens was visible at the bottom of the vitreous.

2. An 80 year old woman with ripe cataract in O. D., V. = 1/300. Iridodonesis nasally and below, probably a defect of the zonule. O. S. V. = 5/50 with + 4 D.; incipient cataract with central venous thrombosis. There was danger of intraocular hemorrhage and consecutive vitreous prolapse, as the venous thrombosis of O. S. made it probable that the vessels in O. D. would not be in the best condition. Two months later the vision in O. S. was reduced to 2/60. An iridectomy upward was done and reclination 2 weeks later. A week later the patient left the hospital with V. = 3/6 and the eye was quiet. Her family physician wrote that the patient saw sufficiently and was satisfied with the result (about one year after operation).

Dubois does not consider the danger of vitreous prolapse (which can reasonably be expected before the extraction), of less importance than that of the reclination. Danger of infection from cyclitis and detachment are equally present, especially when the vitreous prolapse happens before evacuation of the lens. Of the remaining dangers only postoperative glaucoma remains, which may result when vitreous is present in the anterior chamber. Dubois gives little attention to restlessness of the patient as this can be controlled by novocain injections.

He finds the indication for reclination in the following conditions. 1. Cataract in the one remaining eye when the other has been lost by expulsive hemorrhage; 2. An early certain vitreous loss after the corneal incision, on account of a defective zonule, especially if this is the only remaining eye.

Disscussion. Snellen considered a clouded lens after detachment of the retina as one of the indications. He did reclination in such a condition with fair success; the lens rose later somewhat, because he had not disturbed the vitreous. He seldom saw expulsive hemorrhage after extraction and he questioned if the same danger exists for the other eye.

MULOCK HOUWER asked if much fluid escapes, and was the danger for hemorrhage increased?

Dubois stated that no fluid escaped, and the danger for hemorrhage is much less.

WAARDENBURG asked if the lens in its capsule is pressed down and if hypermature cataract was an indication?

Dubois stated that the lens in its capsule is pressed down. Hypermature cataract can show a tremulous iris, but this does not prove a weak zonule; a hypermature cataract is, therefore, no indication for reclination.

ROMER asked in what position the patient is to be operated.

Dubois prefers a reclined position, altho the head may be somewhat higher.

REILING asked if the method of Van der Hoeve with the scleral threads does not give enough security.

VAN DER HOEVE stated that the threads are of no advantage in hemorrhage, and reclination still is indicated.

Accommodation Measure.

NICOLAI described this instrument in 1900, but, altho quite serviceable, it has remained unnoticed. The instrument consists of a small board over which a small carrier can be moved with a lens of 10 D., which is 9.3 cm. from the other end, covered with a cork. Its focal point then coincides nearly with the optical center, if the cork covered end is put against the lower orbital border, vertically below the apex of the cornea. The individual differences are of slight importance. The emmetrope has to relax his entire accommodation to see the target clearly at 10 cm. behind the lens.

One can easily compute, that displacement of the test of 1 cm. represents one diopter of accommodation

The size of the test object is of importance. A normal eye distinguishes 2 black lines as separated, when their distance on the retina is 0.004 mm. The white line which separates these black lines must then have a thickness of 0.025 mm.

Nicolai constructed his test object, or target, as a cross, the one arm having 3, the other 4 black lines, which are separated by white lines 0.025 mm. wide. The refraction is corrected, one eye covered with a frosted glass and the instrument is put against the lower orbital border of the other eye. The target which is 10 cm. behind the lens is moved forward until the patient states that he can no longer separate the black lines. On the measuring tape can be read, in diopters, the accommodation of the patient. In practice, difficulty is sometimes encountered, because the patient can not follow quickly enough, or does not have his attention on it. With less than 6/12 vision the instrument is of no value.

Discussion. ROELOFS has two theoretic objections. 1. As Nicolai corrects the refraction the nodal point of the corrected eye is transposed so that the focal point of the lens of 10 D. does not coincide with it. 2. The distance of the lines. The choice rests on theoretic considerations contrary to practice. Most emmetropes possess a finer distinguishing power. For these, as well as for poorer vision, separation can be taken as a criterion for an exact accommodation.

Recurring Iridocyclitis with Hypopyon.

WEVE gave the anatomic examination of this condition.

Discussion. DUBOIS did not understand why *per se* a connection must be present between the coccisepsis and the ocular condition; he only considers it probable. He asked if cocci were found in the eyes.

VAN DEN BORG inquired as to the primary focus of the infection and if the blood was examined.

WEVE considered the cocci as a cause, because the cases in the literature, where cocci were found, were identical with his. This connection was also accepted on account of the coincidence and succession of the different inflammations. Gilbert examined the blood with negative result. In such acute cases cocci were never found. In the specimens no cocci were found. The primary cause is unknown. The patient had a carbuncle, however, at the age of 16 years.

Treatment of Detachment of the Retina.

E. MARX presented the paper.

Discussion. BRANBERGEN stated that the connection which the speaker traced between the therapeutic action of the saltless diet and the experiments of Hertel on the osmotic changes between blood and ocular fluid are not clear to him. He has repeated the latter and has come to the conclusion that we are dealing chiefly with a physical process, where the artifically disturbed osmotic equilibrium is restored after a few hours. It cannot be stated that a continually acting fluid current could favorably influence dialysis. He reported the results of a conservative treatment (at least 6 weeks in bed and bandage) of spontaneous cases of detachment of the retina, as practiced in the Amsterdam University clinic, during the last ten years.

1. Of 21 cases with visual acuity more than 1/10, three observed 6 months, 1 and 6 years, improved. Five observed 1-2-3-4-4 years, unchanged. Thirteen became worse.

2. Of 21 cases with visual acuity less than 1/10, three observed 6 months, 1, 5 and 3 years, improved. Seven observed 5 months, 1-1-1-1-3-3 years, remained the same. Eleven became worse.

These results are slightly better than most statistics. The results of operative interference were not so good; as only those cases were submitted where rest had failed to give relief. Of the 12 operations evenly divided over trephining with salt injections in the vitreous and the operation of Birch-Hirchfeld, in three cases only there was temporary benefit; never a success which lasted.

MARX remarked that the action of the salt free diet lasts as long as the diet lasts. Disturbance of the equilibrium occurs continuously.

WEVE has used the salt free diet extensively on some thirty patients. He had little success in old cases but in new

cases the result was somewhat better. He considered it especially important before operation, as the fluid is then kept for some time in the tissues.

Atrophy and Marginal Keratitis.

TRESLING reported the case of a 59 year old laborer who came complaining that for the past week he could not stand the light with O. D. The eye was red with conjunctivitis and slight pericorneal injection. In the upper nasal quadrant of the cornea was an infiltration parallel to the limbus, toward which coursed many superficial vessels. A groove was not observed. Downward a point like infiltration was present. The diagnosis of marginal keratitis was made. Vision O. D. = 6/20 not improved with glasses. Vision O. S. with +1 = 6/6.

The eye became somewhat quieter, but the affection progressed along the margin so that some five weeks later, the entire circumference of the cornea was affected. The patient disappeared for half a year but returned, because O. S. also became affected. There was a photophobia with slightly edematous lid borders, especially at the right end on opening the eyes, excessive lacrimation. O. D. showed conjunctivitis and marked pericorneal injection. The cornea seemed smaller and surrounded by a heavily infiltrated, undermined border; which went downward in a grayish, 2 mm. broad, marginal seam. This passed smoothly over into the limbus. The surface of the marginal seam was covered with mucus while many blood vessels ramified over it. There was no epithelial defect. Here and there in this groove were deeper pits somewhat more transparent. At the limbus, the conjunctiva covered, as a thick fold, the margin of the ulcer, while above, in the vertical meridian, a granulation of about 2 mm. was present.

In the beginning, and later over the entire cornea of the right eye, white flocculous precipitates in the parenchyma were seen, with the corneal microscope, similar to the opacities of an arcus senilis. No Descement spots were present but a slight iritis had produced a few synechiae. The corneal sensibility was decreased, the tension was normal and vision, fingers at 2.5 meters. No improvement with lenses.

Left eye. Photophobia. In the upper nasal quadrant, at the same spot as in R., a groove was present along the limbus with the dimensions of an arcus senilis and this ended up and downward in a marginal infiltration of about the same breadth. Its bottom was also mirror like and without epithelial defect. This sector showed episcleral injection. No iritis, no arcus senilis, sensibility normal. Vision with — 1 = 6/6.

The patient had been treated in the hospital since April but without success. In O. D. a small corneal island, 8 mm. diameter, was present, entirely opaque, milky white, surrounded by a light yellow margin, which was higher than the 3 mm. broad marginal zone. It was partly covered with mucus and contained many vessels. Vision reduced to 1/300. The progress in O. S. has been slower, a central corneal portion, 9 mm. in diameter has remained practically clear, but the sensibility is decreasing. The patient remains strongly photophobic in O. D. with a spastic entropion, and he suffers intermittently with severe pains in O. S.

Discussion. LANNS said the case called to mind a similar one, in which an epithelial defect was present and the eye very painful. It could not be benefitted therapeutically. Recently it was rayed with artificial sunlight and showed no progress.

Etiology of Strabismus.

VAN DER HOEVE referred to Roemer's question, "Why do not most people squint?" The static relations of orbits and eyeballs is such, that if the eyes were left to themselves, everyone would squint. To prevent this, normal people have a warning apparatus, which acts continuously, and a correction apparatus which continuously abolishes the ever changing squint. The warning is given by double images; the correction apparatus acts thru the fear of double images which do not enter consciousness. The fear of double images, diplopiaphobia, makes the correction apparatus effective. This apparatus comprises the entire sensomotoric system of the ocular muscles. The double images can disappear because they come together, or by exclusion. For the coming together, the fusion sense is necessary. If it is ab-

sent, the coming together is impossible and exclusion takes place. This can take place in the position in which static strabismus places the eye. It is difficult to disregard double images which are near, so one eye will take a position in which double images are easily excluded, if the eye shuns the double images. To the static squint, an evasion squint is added. The result of these two will be a concomitant strabismus, as the patient will try to keep the double images constantly at the same distance so as to exclude them more easily. People with fusion tendency will try to unite the double image only succeeding if the static squint is slight. If they do not succeed, one of the images must be excluded, which will produce a concomitant strabimus, with or without an evasion squint.

If the coalescence succeeds, the tissues may become accustomed to it if it begins in youth, and the static relation becomes changed so that orthophoria appears. Absolute orthophoria can exist, as the static relations constantly change, consequently these patients need continuous control of the diplopiaphobia and the correction of the sensomotor apparatus.

If no orthophoria appears, then a latent squint (heterophoria) is formed, thru the coalescence under the influence of the continuous correction of the sensomotor apparatus.

This divides patients who, at birth, have a static squint by the presence or absence of diplopiaphobia and fusion tendency, into four groups: orthophoria, heterophoria, concomitant strabismus and static squint.

During the individual development, use of the eyes, refraction conditions, paralysis or cramps of the ocular muscles, etc., play a part. The relation of convergence and positive refraction exists with most people, as Donders taught. If this relation were absolute, every myope and hypermetrope, and every one with the same degree of refraction anomaly in the same measure would squint. This evasion squint explains why patients with the refractive anomaly develop a strabismus with a deviation which is not always like the refraction anomaly. If the double images cannot be brought together the patient will seek a position in which he can neglect most easily the double image. With a refraction squint, an evasion squint develops so that a combination squint appears, not conformable to the refraction, and concomitant squint results. Correction of the refraction dispels this and the evasion squint, when they have not existed too long. A persistence of too long standing accustoms the tissue to the faulty position and the refraction can correct the squint only partially. The squint, which remains after correction, combines with new evasion squint, if the exclusion does not succeed.

Discussion. BRANBERGEN asked if the double vision of seeing two things in one direction causes trouble; and Van der Hoeve replied that both conditions produce trouble.

ROELOFS could not accept the entire theory. He cannot consider strabismus as an evasion squint. It is true that the deviation often is larger than agrees with the slight hyperopia; he considers that the infant, when it begins to squint, converges strongly, because it accommodates with difficulty. The accommodation then follows the convergence and not the reverse. In incipient strabismus, when the deviation is still periodic, it appears chiefly when the child is tired or intensely busy with something. He does not understand why an evasion squint should appear. He can imagine very well that the wish for sharp images in accommodation causes a continuance of the convergence innervation; this convergence innervation has not the slightest hold with an evasion strabismus.

VAN DER HOEVE does not believe that a tired child will converge rather for an exact accommodation than for avoiding double images; or to maintain the convergence innervation. Not all concomitant strabismus is evasion squint.

WAARDENBURG asked if this strabismus should be considered a supranuclear regulation.

VAN DER HOEVE considered in his series B (with static squint) all people with congenital absence of diplopiaphobia, a deviation therefore of the consciousness. Secondarily an absence of diplopiaphobia may originate thru one of the images being of lesser value, as

in congenital amblyopia, and acquired visual inferiority thru different causes. He thinks evasion in the horizontal meridian the easiest escape of double images; however, evasion in vertical direction is also seen.

Observations and Experiments Regarding Corneal Ring Abscess.

H. F. FLIERINGA observed a case and made some experiments on rabbits eyes to elucidate its causative factor. A piece of iron was extracted from the eye of a smith a few hours after the accident. Thru lack of cooperation of the patient and boring of the piece of iron into the iris, the latter became detached from its root for the greatest part and had to be partially excised. The following night there was some pain and the next morning there was chemosis, injection, etc. During the next few days, about 1 mm. from the limbus in the cornea, a slight grayish ring shaped opacity appeared. This opacity was 1 mm. broad, increased in width and became yellowish, resulting in a typical annular abscess. The exudate around the perforation yielded pure cultures of staphylococcus aureus. From the partly purulent vitreous, only bacillus subtilis could be cultivated. A connection between the secondary trauma and the annular abscess was thought of, and experiments led to the following conclusions: The annular abscess was not a separate pathologic picture, but a symptom of a grave infection of the inner eye. The formation of the annular abscess was caused most probably thru penetration of microorganisms or toxins into the cornea. They come from the anterior chamber or vitreous cavity in the neighborhood. Their formation is favored by traumatism in the neighborhood of the angle of the anterior chamber as tearing off of the entire iris, iridodialysis, inner scleral ruptures, producing rents, which allow vitreous and aqueous entrance into the tissue spaces of the cornea. It is unknown why the annular abscess leaves the peripheral cornea untouched; perhaps a better nutrition protects against the noxious action. Moderate increase in tension favors the formation of the annular abscess, as this presses the noxious agents into the cornea. Marked increase of tension obstructs the formation, as this presses the corneal spaces together.

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The following list includes the names of ophthalmologists who have received the certificate of the Board. It will be found convenient in referring patients to a competent oculist in other parts of the country, particularly in the case of younger men and those living in smaller cities who have not become generally known to the profession thru teaching or hospital connections or in other ways.

Adams, Charles F.....Trenton, N. J.
Allen, Thomas Dyer.....Chicago, Ill.
Allport, Frank.....Chicago, Ill.
Alvis, Bennett Y.....St. Louis, Mo.
Andrews, Joseph A.....Santa Barbara, Cal.
Appleman, Leighton F.....Philadelphia, Pa.
Atkinson, Walter S.....Watertown, N. Y.
Baer, Benjamin F., Jr.....Philadelphia, Pa.
Bahn, Charles A.....New Orleans, La.
Ball, James Moores.....St. Louis, Mo.
Ball, M. W.....Warren, Pa.
Bankes, Claude W.....Reading, Pa.
Barber, Frank.....Rochester, N. Y.
Barkan, Hans .....San Francisco, Cal.
Barnes, J. Steele.....Milwaukee, Wis.
Beach, Bennett S.....New York City
Beach, Sylvester J.....Augusta, Maine
Bedell, Arthur J.....Albany, N. Y.
Beideman, Jos. E.....Norristown, Pa.
Bell, Mace Hudson.....Vicksburg, Miss.
Benedict, Wm. L.....Rochester, Minn.
Bennett, Arthur G.....Buffalo, N. Y.
Berens, Conrad, Jr.....New York City
Berrisford, Paul Dee.....St. Paul, Minn.
Binger, Henry E.....St. Paul, Minn.
Black, Melville.....Denver, Colo.
Black, Nelson.....Milwaukee, Wis.
Blair, Wm. W.....Pittsburgh, Pa.
Blake, Eugene M.....New Haven, Conn.
Blue, J. B.....Memphis, Tenn.
Boerner, Morris H.....Austin, Texas
Bordley, James, Jr.....Baltimore, Md.
Briggs, H. H.....Asheville, N. C.
Brinkerhoff, N. M.....Philadelphia, Pa.
Brooks, Earl Brisbin.....Lincoln, Nebr.
Brown, Alexander.....San Francisco, Cal.
Brown, John Edwin.....Columbus, Ohio
Brown, Samuel H.....Philadelphia, Pa.
Brownell, Morton T.....Oneonta, N. Y.
Brose, Louis D.....Evansville, Ind.
Bruner, Abram B.....Cleveland, Ohio
Bruner, William E.....Cleveland, Ohio
Bruns, Henry Dickson.....New Orleans, La.
Buchanan, Mary.....Philadelphia, Pa.
Buck, Robert H.....Chicago, Ill.
Bulson, A. E., Jr.....Ft. Wayne, Ind.
Burch, Frank E.....St. Paul, Minn.
Butler, W. K.....Washington, D. C.

Buvinger, Chas. W.....East Orange, N. J.
Cadwell, Chester T.....Poughkeepsie, N. Y.
Calhoun, F. P.....Atlanta, Ga.
Calhoun, James G.....St. Louis, Mo.
Campbell, Don M.....Detroit, Mich.
Carpenter, Ernest W.....Greenville, S. C.
Carson, W. E.....Pittsburg, Pa.
Carvill, Lizzie M.....Boston, Mass.
Cayce, Eldred B.....Nashville, Tenn.
Chance, Burton.....Philadelphia, Pa.
Charles, Jos. W.....St. Louis, Mo.
Chapman, Vernon A.....Milwaukee, Wis.
Cheney, Fred. Edwin.....Boston, Mass.
Clapp, Clyde Alvin.....Baltimore, Md.
Clark, Charles F.....Columbus, Ohio
Clark, Frederick T.....Westfield, Mass.
Clark, Ivor G.....Columbus, Ohio
Clement, Charles G.....Chicago, Ill.
Cohen, Martin.....New York City
Conlon, Frank A.....Lawrence, Mass.
Connole, Jos. V.....Philadelphia, Pa.
Cook, W. Albert.....Tulsa, Okla.
Copps, Lyman Alden.....Marshfield, Wis.
Cordes, Frederick C.....San Francisco, Calif.
Cowan, Alfred.....Philadelphia, Pa.
Crebbin, Alexander R.....New Orleans, La.
Crisp, Wm. H.....Denver, Colo.
Crockett, Robert L.....Oneida, N. Y.
Cross, George H.....Chester, Pa.
Culver, Chas. M.....Albany, N. Y.
Cunningham, Allan R.....Halifax, N. S.
Curdy, Robert J.....Kansas City, Mo.
Cushman, Beulah.....Chicago, Ill.
Dancy, A. B.....Jackson, Tenn.
Darling, Chas. G.....Chicago, Ill.
Davidson, Herman P.....Chicago, Ill.
Davis, Wm. Thornwall.....Washington, D. C.
Dean, Frank Wilson.....Council Bluffs, Ia.
Dean, Alfred.....Grand Rapids, Mich.
Dennis, D. N.....Erie, Pa.
Dennis, Edw. P.....Erie, Pa.
Derby, George S.....Boston, Mass.
Dickson, John F.....Portland, Ore.
Dimitry, Theodore J.....New Orleans, La.
Donovan, John A.....Butte, Mont.
Dorge, Richard I.....Minneapolis, Minn.
Dorsey, J. G.....Wichita, Kansas
Dowling, J. Ivimey.....Albany, N. Y.
Downey, Jesse W., Jr.....Baltimore, Md.
Duckworth, Guilford, M.....Cuero, Texas
Duncan, Jos. L.....Pittsburgh, Pa.
Dunlap, Lawrence G.....Anaconda, Mont.
Dunnington, John H.....New York City
Easton, Elwood T.....Boston, Mass.
Ellegood, Joshua A.....Wilmington, Del.
Ellett, Edward C.....Memphis, Tenn.
Ellis, Edward K.....Boston, Mass.
Elwood, Calvin R.....Menominee, Mich.
Emerson, Linn.....Orange, N. J.
Esterly, Daniel E. (deceased)..Topeka, Kans.
Evans, John Norris.....Brooklyn, N. Y.
Ewing, Arthur E.....St. Louis, Mo.
Fagin, Robert.....Memphis, Tenn.
Fairchild, Nora M.....Omaha, Nebr.
Fairing, John W.....Greensburg, Pa.
Feingold, M.....New Orleans, La.
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American Journal of Ophthalmology

Series 3, Vol. 8, No. 1 January, 1925

PUBLISHED MONTHLY BY THE OPHTHALMIC PUBLISHING COMPANY

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Copy of advertisements must be sent to the Manager by the fifteenth of the month preceding its appearance.

The annual subscription for the JOURNAL and the OPHTHALMIC YEAR BOOK is eleven dollars, in advance.

Subscriptions, applications for single copies, communications with reference to advertising or other business, should be addressed to the Manager of Subscriptions and Advertising.

JEAN MATTESON, ROOM 1209, 7 West Madison Street, Chicago, Ill.

PREPARING DIABETICS FOR OPERATION.

The diabetic patient is not considered a good subject for operation. How much the risk of an unfavorable result is generally increased in diabetes is a matter of debate; and the seriousness of the danger in the individual case is hard to estimate. But in any case, it should be reduced to a minimum, if time is available for preoperative treatment. The arteriosclerosis present in cases of diabetic gangrene may account for many bad results recorded for operations on diabetics; but something can be blamed on impaired nutrition and lessened immunity toward postoperative infection.

The most formidable menace, however, is the danger of postoperative coma and death thru acidosis. This danger is increased by use of a general anesthetic. But it is also increased by mental and emotional excitement, the perturbations that attend any operation which has been dreaded, or from which a great deal is hoped, and performed under local anesthesia when the patient is necessarily conscious of what is going on and making a strong effort to cooperate. These latter conditions are present, often causing a high degree of emotional stress, in cases of cataract extraction, the most common operation of first importance that the oculist is called upon to do on a diabetic. There is commonly no reason why such an operation may not be deferred until the patient is brought into the best condition of which he is capable.

Of late years the diatetic treatment of diabetes has been brought to a high degree of efficiency; and now insulin gives, at least for the time, almost complete control of the disease. The first point in dealing with a diabetic, or suspected diabetic, who needs an operation like removal of a cataract, is to have the sugar content of his blood carefully estimated. Normal blood contains about one-tenth of one per cent of sugar. If this is much increased, doubled or nearly so, the patient is to be treated as a diabetic, even tho no sugar is found in the urine. Probably the danger of diabetes is more nearly proportioned to the sugar in the blood than to the amount in the urine, altho mostly the proportion increases together in both. Increased blood sugar may be found in any patient with senile cataract, altho not suspected of diabetes. In such cases it is as important to examine the blood for sugar

as the urine for albumin and casts.

If sugar is found in the urine, or an excess of sugar in the blood, the effort should be made to get rid of it, or reduce it to a minimum, with diet, and small doses of insulin. When the urine becomes sugar free, or the blood sugar falls to the normal proportion, the diet may be made more liberal until the patient ceases to lose weight. When this is accomplished, without any return or increase in sugar, the proper condition for operation has been reached.

When it is not possible to thus eliminate all sugar excess, the danger of "diabetic coma," really coma from acidosis, can be avoided by bringing the patient into an improved condition and then using insulin by this method: During the preparation immediately before the operation, give the patient a large dose of glucose and enough insulin to secure its oxidation. In this way the danger of postoperative coma and death is eliminated. In such cases the ideal thing is for the ophthalmologist to have the assistance of an internist, who is working with the diabetic treatment of diabetes and insulin. But with careful reading of the recent literature on these subjects any well trained practitioner can undertake such a course of treatment, and give his patient a much better chance of a good result from the operation, than he would have without such preparation. E. J.

THE LONDON CONVENTION.

Those wishing to become members of the Convention of English Speaking Ophthalmological Societies should send the registration fee as soon as possible to the Secretary of the Finance and Registration Committee, Mr. Leslie Paton, 29 Harley Street, W. 1, London, England. Official invitations have been received by the officers of the Section on Ophthalmology of the American Medical Asociation, The American Ophthalmological Society, and The American Academy of Ophthalmology and Oto-Laryngology.

The invitation covers all members of these organizations. In the case of the Section, this will cover all interested members of the American Medical Association since any member may join the Section by so registering at any annual meeting. The registration fee is two pounds, and for ladies accompanying members of the Convention, who wish to become Associate Members, it is ten shillings. Each member of the Convention will receive a copy of the transactions when published.

A preliminary bulletin will shortly be issued; but certain portions of the program are now announced.

The following selected speakers will take part in a symposium "On the evolution of binocular vision": Sir Charles Sherrington, F. R. S.; Sir Arthur Keith, F. R. S.; Sir Frederick Mott, F. R. S.; Prof. Elliot Smith, F. R. S.; Prof. S. E. Whitnall, (Montreal).

The discussion on "Microscopy of the living eye" will be opened by Dr. Gordon Byers (Montreal); Mr. Harrison Butler (Birmingham); Dr. Arthur J. Bedell (Albany); and Mr. Basil Graves (London).

Those wishing to take part in this discussion, to read papers, to give demonstrations, or to contribute exhibits to the Museum, are requested to communicate with the Secretary of the Scientific Business Committee, Mr. R. Foster Moore, 91 Harley Street, London, W., England.

On the evening of Monday, July 13th, before the opening of the Convention, a reception will be held at the Royal College of Surgeons of England. On Saturday, July 18th, at the conclusion of the Convention, there will be an excursion to Oxford and to Cambridge. E. J.

GRADUATE COURSES.

The increase of opportunities to study ophthalmology, as the graduate in medicine wishes to study it when preparing for special practice, or keeping up with its advances, is extremely gratifying. To have such opportunities is a pleasure and a stimulus to every specialist who appreciates how much is to be learned that will help to make him more skilful and more

successful in his professional work. The arrangement and announcement of such courses as are noticed in this issue under "News Items" implies a wider interest and demand for them.

The Graduate School of the University of Pennsylvania shows faith in the awakened interest of American ophthalmologists regarding microscopy of the living eye, by bringing Mr. Basil Graves from London to give a series of two weeks' courses upon it. Each class will be limited to six students and each student will thus have the benefit of personal instruction. Mr. Graves, who has devoted his year's incumbency of the Lang Scholarship to the study of this subject, also worked with Vogt at Zurich. He has contributed observations on a new extension of this kind of microscopy in this Journal, and has also written a paper on the apparatus required for it, that will follow.

While it is logical to suppose that only those who buy the apparatus and use it can get the full benefit of such instruction, no ophthalmologist can take such a course without having his ideas of scientific ophthalmology extended and enriched by a new conception of the value of exact observation. While this course only occupies two hours each day, the ophthalmic clinics and the medical libraries of Philadelphia will certainly make it possible for students to fill their whole days with profitable ophthalmic work.

The University of Colorado announces that its three months graduate course in ophthalmology, heretofore given during the summer, will hereafter be given during the winter and spring. This has been rendered possible by the opening of the new medical school and hospital at Denver, where the conditions for teaching now seem ideal, as compared with those of the last twelve years under which this course developed and won its reputation. As heretofore, condidates for the special degree will be required to show their special preliminary work in mathematics, do the additional year of clinical work and present an original thesis.

The third intensive two weeks' summer course on ophthalmology and otolaryngology will be given in Denver, this year August 10 to 22 inclusive. As regards ophthalmology, the leading topics will be microscopy of the living eye, pathology and operative technic. Short courses like this meet the needs of those who already have a good, general acquaintance with ophthalmology, and desire to continue developing their professional knowledge and skill, mastering new ideas and new methods of proved value; and thus gain and hold advanced rank in the army of ophthalmologists.

An equally important function is performed by schools that offer courses extending over a full year or more, in preparation for entering upon ophthalmic practice. To such schools we must look more and more for the training of the ophthalmologist of the future. The haphazard method of each aspiring specialist, studying what he thought best, as much as he felt inclined to do, without advice or guidance, and choosing a school under the influence of the advertising, never was a good method, and must rapidly become unpopular under the standardized requirements of the American Board for Ophthalmic Examinations.

There are several institutions that are presenting good opportunities for small numbers of students able and determined to spend years in preparation for ophthalmic practice; and now equal opportunities are being opened to larger numbers by systematic courses including work in fundamental branches, like anatomy, histology, physiology and pathology of the eye and related parts, with abundant clinical work for those who have prepared to profit by it. Such a complete course of one year has been offered for four years by the University of Pennsylvania, and is now offered by the Chicago Postgraduate Hospital and Medical School, which utilizes the great ophthalmic clinical service of the Cook County Hospital.

In Vienna, the Minerva Book Store has published an account of the graduate courses offered in the Allgemeines Krankenhaus and in the two Eye Clinics. But this refers to the Ameri-

can Medical Association of Vienna, as the source for detailed information with regard to the courses given in English. We note that the special intensive two months course given in English the last two years was not given in 1924. Perhaps the difficulties of arranging such a course, dependent on the full cooperation of the English speaking teachers in both clinics, was greater than those outside could appreciate. E. J.

BOOK NOTICES.

Comunicaciones del Hospital Oftalmológico, Buenos Aires, 1923. Paper, 107 pages, 1 colored plate, 17 illustrations.

This annual Report of the Ophthalmological Hospital, is the sixth published, and contains seven original papers, a resumé of the monthly meetings of the Ophthalmological Society of Buenos Aires and some abstracts. The first article describes the opening of the new building of the Hospital, in a suburban part of the City, where the bed capacity has been increased to 120. During the year 1922, 22,852 new patients were recorded, 2,258 operations performed, and between 500 to 600 patients examined daily.

Besides the Ophthalmic Services, the Laboratory and Pharmacy, new Departments of radiology, medical and surgical clinics, oto-rhino-laryngology and dermatology will be opened as annexes to the hospital, for the complete examination and treatment of the patients.

The original articles in the journal are: *Colored vision and its anomalies* by Dr. F. J. Soriano; an exhaustive review of the subject, and critique of the many theories advanced for explaining the physiology of vision and its pathologic changes.

Drs. Marque and Oyenard. *A case of amaurotic family idiocy*, in a child, a son of a Russian Jew immigrant. This disease is very rare in Buenos Aires.

Dr. A. Oyenard. *Frequency of trachoma in Buenos Aires.* The records of the Ophthalmological Hospital during the last 15 years, for 200,000 patients observed, show a great decrease of trachoma. In the year 1908 the proportion was 9.6%, while in 1923 it came down to 2.3%. The average of these 15 years is 3.9%.

This marked reduction must in part be attributed to the world war, which cut down the amount of the immigration considerably. Before 1914 the percentage was 7.7%, and gradually came down to 2.2% in 1922, with a new slight increase in 1923-24. Good hygienic surroundings, undoubtedly superior in Buenos Aires to those of the original countries of the immigrants, may also account for the checking of the infection in the city.

Dr. L. M. Echavarria. *Ocular complications of sinusitis.* Report of two cases of frontal and maxillary sinusitis with optic nerve involvement.

Dr. E. Adrogué. *Alcohol and tobacco amblyopias.* The author calls attention to the increase of these diseases in Buenos Aires. Of 205 cases of optic nerve diseases, 48 were due to toxic causes. That gives for 23,633 new patients in the year 1922, the proportion of 0.2%; still inferior to 0.5% found by Sattler in the Koenigsberg Clinic.

Adrogué considers the tobacco poisoning as the principal factor in his cases, the alcohol habit being a predisposing cause, and advocates barring from the market the tobaccos of inferior quality, when the percentage of nicotine goes above certain limits; and also when the moisture they contain is higher, preventing a perfect combustion.

Dr. E. Adrogué. *Cholesterin crystals in the anterior chamber.* In an eye affected with complicated cataract, posterior synechiae and atrophy of the iris, the anterior chamber was filled with numberless small crystals which strongly reflected the light. With the slit lamp these crystals show the circulation of the aqueous perfectly. There is a current behind the cornea moving downward, and another near the anterior surface of the iris going upward. Puncture of the anterior chamber showed the crystals to be formed of cholesterin.

Dr. E. Adrogué. *Retinitis pigmentosa without pigment.* Man 28 years old with hemeralopia and great concentric contraction of the fields. With the ophthalmoscope the retina appears gray all over

the fundus; two or three irregular pigmented spots showing in the periphery; and postretinitic atrophy of the papilla, with thread like vessels. With red free light the picture is very interesting. The retina has a well defined, atrophic, grayish white aspect from the periphery to the center, with small spots of a darker color and small whitish reflexes. Around the macula there are other more characteristic reflexes due to folds of the internal limiting membrane of the retina, shown in an interesting figure. These folds disappear in the macular region in which there are isolated irregular reflexes. The fovea is increased in size and has a peculiar granular appearance, due probably to a beginning cystoid degeneration.

M. U. T.

Handbuch der biologischen Arbeitsmethoden. Edited by Geh. Med.-Rat. Prof. Dr. **Emil Abderhalden.** Abt. V. Methods of Examining the Functions of the Various Organs of the Animal Organism. Paper, 280 pages, 172 illustrations, 5 tables. Urban and Schwarzenberg, Berlin and Vienna, 1924.

I. **Refraction, Visual Acuity, Accommodation and Anomalies of Refraction of the Eyes. C. A Hegner,** Lucerne. 118 pages.

II. **Determination of Refraction. Hugo Wolff,** Berlin. 14 pages.

III. **The Course of Rays in the Eye and in Medico-Optical Instruments. Moritz von Rohr,** Jena, 148 pages.

These three sections are of the Handbook form, Lieferung 138, and are part of the volume on the Light Sense and the Eye, of which three parts have already appeared. For a proper understanding of the present one, a knowledge of German must be supplemented by a good working knowledge of higher mathematics. This is especially true of I and III. For those so equipped, this publication will prove of the greatest value, especially since the numerous illustrations aid materially in the understanding of these complicated subjects. Part II is not, as its title would seem to indicate, a description of methods of testing the eye, but instead deals with only a part, the objective refraction. The greater part is devoted to skiascopy, and 5 tables, 4 colored, show the course of the rays during this method of examination.

C. L.

Lighting in Relation to Public Health. Janet Howell Clark, Associate Professor of Physiological Hygiene and Public Health of the Johns Hopkins University. 192 pages, 67 illustrations, 38 tables. Cloth. Published by Williams and Wilkins Co., Baltimore, Md., 1924.

The author states that the book gives, in a condensed form, the subject matter of a course which she has been giving at the Johns Hopkins University. It is a nontechnical, but scientific discussion of an important factor in the school, hospital, factory, office, store and on the public highway. There are 15 chapters, as follows: I. Units and Standards, 5 pages; II. The Measurement of Illumination, 6 pages; III. The Development of Illumination, 11 pages; IV. Reflectors, 19 pages; V. Sufficient Illumination, 13 pages; VI. Glare, 11 pages; VII. Best Conditions for Visual Efficiency, 8 pages; VIII. Lighting of Schools, 10 pages; IX. Factory Lighting, 19 pages; X. Other Interior Illumination, 6 pages; XI. Exterior Lighting, 8 pages; XII. Occupational Eye Diseases from Low Illumination, 9 pages; XIII. Pathologic Effects of Radiant Energy on the Eye, 12 pages; XIV. Occupational Eye Diseases Attributed to Excessive Light and Heat, 15 pages; XV. Protective Glasses and Glasses for Refractive Errors, 8 pages.

Oculists continually preach more proper illumination and protection of eyes of pupils and workers, but how many, if suddenly called upon, could furnish the necessary information to carry out their recommendations—or for that matter could furnish the recommendations in a concise, workable form? Failing that knowledge from individual study, to what source can he go for the information? With the publication of the present book, the latter

question, at least, is answered. Even then, the searcher will find it necessary to do some hard studying, but the results will repay him for his labor.

The majority of oculists will be most interested in Chapters VII, and XII—XV inclusive. Quoting a few of the points brought out: "The eye fatigues least under the following systems in order of preference (a) total indirect, (b) semiindirect with dense globe, (c) direct with filament hidden and brilliant lining or reflector dulled at the edge to reduce the glare." "The development of myopia..... is attributed as a rule, to a combination of hereditary tendency and overuse of the eye, the strain from overuse being greater when the eye is used in a poor light." "Wave lengths shorter than 313$\mu\mu$ are completely absorbed by the eye media before they reach the retina, and wave lengths between 400 and 313$\mu\mu$ are partly absorbed, and the absorption of this energy in the eye media results in photoelectric and photochemical changes which are apt to be injurious to the eye." "Wave lengths greater than 760$\mu\mu$ (infrared) do not produce vision, and when they are absorbed by the eye media, the energy is transformed into heat." "There is no evidence of immediate and direct injury to the eye from heat, except in eclipse blindness." The diseases attributed to light and heat are cataract and eye trouble in the motion picture industry. The work concludes with a discussion of protective glasses for ultraviolet rays, for visible light, and for infrared rays.

C. L.

The Ophthalmic Year Book, Volume XX. Edited by Edward Jackson and William H. Crisp, with the assistance of 34 collaborators. Cloth, 433 pages, 17 illustrations. Chicago, The Ophthalmic Publishing Co.

In 1899, Lea Brothers & Co., of Philadelphia, started a four volume, annual review of medicine and surgery, under the name of "Progressive Medicine." In the second volume of each year, 75 pages were assigned to ophthalmology. Because the interest of the general medical profession in ophthalmology did not seem to justify giving it more, in 1904, the space given for this part of the work was cut to 25 pages, the support received not meeting the outlay required by the original plan.

Five years reviewing of the world's literature relating to our specialty had shown the great value, to the specialist, of having such literature somewhat at his command. No acquaintance with text-books, no training in what had been the general knowledge and practice of the specialty, could possibly takes its place. To give opportunity for such acquaintance with the literature, the Ophthalmic Year Book was started and has been kept up. The financial cost has always been greater than the receipts from the book. But the editors and collaborators have had the advantage of their own better knowledge of the literature, to compensate for the time and labor entailed in preparing such a work.

The first volume, in 1904, consisted of 260 pages. The size of page was enlarged and the number grew, until Volume VIII, 1911, had 455 pages. Then an interval of eighteen months ensued between issues, and Volume IX, reviewing the literature of nearly two years, had 518 pages. For this volume, there were nine collaborators. The subscription price was repeatedly increased, but still left a deficit for actual printing and binding of the volume. From 1912 to 1917, the Year Book received important financial assistance from the Knapp Memorial Fund, of the Section on Ophthalmology of the American Medical Association. Its size reached the maximum of 545 pages in 1915.

With the consolidation of American ophthalmic publications, to form the AMERICAN JOURNAL OF OPHTHALMOLOGY, the Year Book page was enlarged to conform to that of the new journal; and the Year Book appeared as a supplement, paged independently, of the JOURNAL. In 1919, the Year book was published as a periodical, in quarterly parts; and, to gain admission to the mails as a periodical, had to be given the name "Ophthalmic Literature."

However, in 1923, the desire of subscribers to have it in the form of a bound, annual volume, led to the resumption of nearly its original form, except that the larger journal page has been continued.

This sketch of its history seems appropriate, now that 20 volumes, covering 21 years of the literature have been issued; and the editorial management has passed to the hands of Dr. Crisp; the former editor taking, what he hopes will be a useful place, among the collaborators.

It is within the bounds of moderation to say that the present volume is the best that has yet been issued. There is a better selection of matter to be used, there is less that will in a few years be proved of little value and properly forgotten. The survey of the world's literature has been more complete; the different sections have been more carefully prepared for publication. Partly, this is due to more and better working collaborators; partly to the expenditure of more time and energy by the new editor, than was possible for his predecessor.

Improved quality in the work must compensate for the delay in its appearance. No one, not engaged in editing such a publication, can imagine the number of delays that may arise. They begin with the difficulties of securing the publications to be reviewed by the collaborators, accumulate thru the inability of the collaborators and editor to exclude all other matters that claim their time and attention, appear in delays of mails, composing room, proof reading, press work and binding. It is to be hoped that persistent effort will reduce them, so that in the future the volume can be issued near the middle of each year. So long as it continues to come, its appearance will be welcome. E. J.

Die Krankheiten der Augenlider. Ludwig Schreiber, Professor in Heidelberg. Paper, 618 pages, 139 illustrations. Berlin, Julius Springer, 1924.

This book is published as a part of the third edition of the Graefe-Saemisch "Handbuch der gesamten Augenheilkunde" under the editorial supervision of Th. Axenfeld and A. Elschnig. It is based on the work of J. Michel, which constituted Chapter 4 of Volume 4 of the original "Handbuch." But it has been well revised and is the most up-to-date and complete monograph upon its subject that now exists in any language.

It is arranged in four parts: A. Diseases of the Skin of the Lids, 383 pages; B. Diseases of the Tarsus, 47 pages; C. Diseases of the Muscles and Nerves, 120 pages; D. Position, Form, Size and Variations of the Palpebral Fissure, 40 pages. The completeness of the work may be appreciated by giving the list of headings in part: A. I. Disturbance of Circulation. II. Inflammations. III. Abnormalities (Hyperkeratosis and Parakeratosis). IV. Hypertrophy. V. Atrophies and Degenerations. VI. Dystrophy. VII. Pigment Anomalies. VIII. True Tumors (Blastoma). IX. Dermatomycoses. X. Protozoan Diseases. XI. Diseases of the Glands and Cilia of the Lids. Under each of the headings are subheadings, giving systematic arrangement to the large number of conditions considered.

This systematic arrangement, with a full table of contents, makes it rather easy to locate the part of the book in which any particular condition is likely to be mentioned. In this volume, there are also good alphabetic indexes of subjects and authors' names. There are almost no apparent omissions of what might have been included. Even border-line topics are mentioned, that in a complete system of medical literature would be classed under general conditions; such as trichina and other parasites, myasthenia gravis, favus, etc. The subject of blastomycosis of the lids has never received the attention in Germany that its practical importance in some parts of the world would justify. But in this book it receives a valuable if still inadequate notice.

At the conclusion of each topic, a bibliography is appended. These bibliographies aggregate over 65 pages and constitute an extremely valuable part of the book. They include over 2,000 titles; and the names of the

authors here mentioned are not included in the index of authors' names which gives only those whose observations and views have been quoted in the text. The bibliographies are arranged chronologically; a paragraph being given to each year in which some paper has been published on that particular subject.

The book is illustrated chiefly with reproductions of photographs of cases and microscopic sections illustrating the pathology of the conditions described. Of the illustrations, 36 are in colors; and 42 are new, in this edition, having been supplied by cases from the University Eye Clinics at Heidelberg and Prague. They are not collected on plates away from the text relating to them, but are all printed on the same pages with the letter press relating to the case or condition illustrated.

Schreiber's book will be welcome to all German reading ophthalmologists. It well sustains the reputation of the Graefe-Saemisch "Handbook" as the greatest collection of monographs relating to ophthalmology to be found in any language. E. J.

CORRESPONDENCE.

Test for Abducens Paralysis.

To the Editor: While listening to Dr. Gifford's interesting paper at the Montreal meeting of the Academy, I was reminded of a point in connection with the diagnosis of paralysis of the external rectus muscle in small children that has been of value to me. I did not mention it in discussion because I thought it was probably familiar to the Fellows, but after the session I did speak of it to several to whom it did not seem familiar and a short note of it is probably worth printing.

It has occurred to me a number of times in examining a small child with a convergent squint that I was unable to get the patient to turn the eye as desired, and therefore it was difficult to tell whether or not the condition was the ordinary concomitant squint or a paralysis of the external rectus. In such circumstances the function of the muscle can readily be investigated by applying the caloric test for functional activity of the labyrinth. If the ear on the side to be tested is douched with either hot or cold water, a lively nystagmus is excited, provided of course that the muscles are capable of functioning. The test is conclusive, easily applied and involves no question of cooperation on the part of the patient. Its limitations are more theoretic than real, for while destruction of the labyrinth would invalidate the test, in the cases in which it is desired to apply it for this purpose the labyrinth would rarely happen to be dead.

The caloric test and its resultant nystagmus are of course perfectly familiar to every one. It is this particular use which can be made of the test that may not be so generally known.

E. C. Ellett.

Memphis, Tenn.

Angioma of Orbit. Electrocoagulation.

To the Editor:

The additional data and remarks regarding the case published last month in the A. J. O., Vol. 7, p. 946, will add to its interest.

An additional pathologic report has been made by Dr. Eugene J. Asnis as follows:

Growth in orbit consisting of brownish black irregular masses varying in size one cm. to one and a half cm. Portion of the growth appeared dense and calcareous.

Histology in detail: The histologic picture presented a dense matrix of connective tissue; embedded within were numerous dilated blood vessels. The dilated veins varied in size, some being small, others enormously distended. Some of the vessels were filled with thrombi, the latter showing various stages of organization and calcification. While the major portion of the growth is of a simple cavernous angioma type, there are evidences in certain areas of the microscopic field of proliferation of the endothelial cells.

Diagnosis: Cavernous angioma showing tendencies to endothelial proliferation. (Cavernous angioendothelioma).

In view of the fact that radium has been utilized occasionally in angiomatous degeneration of the orbital blood vessels, I have asked Dr. Wm. L.

Clark to permit me to add to the above report his reasons for prefering electrocoagulation and I herewith append Dr. Clark's reply.

"Electrocoagulation was used in this case in preference to radium for the following reasons.

1. After comparative studies covering a period of many years electrothermic methods have been found more efficacious than radium in large cavernous angiomata.

2. The malignant changes as reported by a most capable pathologist indicated that a radical procedure should be chosen and the electrocoagulation method fulfilled this requirement.

3. The patient was suffering much pain, that the reaction following radium treatment would increase rather than diminish. The pain is relieved by coagulation.

4. Radium in sufficient dosage to influence the lesion, would undoubtedly cause necrosis of the orbital bones. This is entirely avoided by the electrocoagulation method if the operation is performed correctly.

5. The fibrosis resulting from radium treatment would cause great pain for an indefinite period, and recurrence of the malignancy would be more likely than following electrocoagulation.

6. The result has justified the means, and radium certainly could not be depended upon to accomplish such a result.

7. As one of the pioneers in the use of radium, which has been found to be invaluable in many lesions, I can definitely recommend electrocoagulation in preference to it in orbital angiomata, or in any condition where complete exenteration is indicated."

G. Oram Ring.

Philadelphia, Pa.

Corneal Dystrophy with Diabetes.

To the Editor:

In the October, 1924, number of The American Journal of Ophthalomology, on page 762, I am quoted by Dr. Lloyd B. Whitham, of Baltimore, Maryland, on "Dystrophy of Cornea and Diabetes" as making in Case 2, W. M. H. a diagnosis "fibrous ulcer of the cornea." This statement was probably given Dr. Whitham by the patient, and is as reliable as the usual report by a patient of one physician's statement during consultation with another physician. My diagnosis was kerato-irido-cyclitis, and seemed to be borne out by the distress of the patient from the photophobic, lacrimating left eye, the lids of which were swollen; conjunctiva, both palpebral and bulbar, markedly inflamed, marked circumciliary injection; cornea hazy and entire structure involved, its anterior surface roughened and oily in appearance, and well marked deposits on Descemet's membrane; pupil irregularly dilated by the atropin solution in use by the patient when he consulted me; tension to fingers soft. A urinalysis disclosed sugar six per cent, acetone slight excess; diacetic acid, none; albumin, trace; hyalin casts, few. The dental radiographs were reported to show alveolar absorption of all teeth, and periapical absorption of lower right second bicuspid. A note on my record card states "good deal of shadow all around and along tooth" extracted.

I feel that my request to have the misquotation corrected is in line with your endeavor to publish a journal of help and interest to us all, containing statements of scientific fact.

Calvin E. Williams.

New York City.

(Dr. L. B. Whitham writes:

"The diagnosis 'fibrous ulcer of the cornea' was simply a part of the patient's history, *as given by himself* and was, for this reason, purposely put in quotation marks. In fact his whole history was so naïvely described that I tried to adhere, as closely as possible, to his actual wordin. It appealed to my sense of humor, particularly when he consulted another oculist who advised enucleation and was so frightened that he never returned.

"Often in my own experience, after having taken great pains to describe and explain matters to my patients, I have been 'flabbergasted' to hear that they have quoted me quite ignorantly and incorrectly.")

ABSTRACT DEPARTMENT

Reprints and journal articles to be abstracted should be sent to Dr. Lawrence T. Post 520 Metropolitan Building, St. Louis, Mo. Only important papers will be used in this department, others of interest will be noticed in the Ophthalmic Year Book.

Maddox, Ernest E. Arrow Test for Astigmatism. Brit. J. Ophth. v. 8, 1924, p. 318.

The arrow makes an excellent test object, when printed on a rotating disc, for locating the axis of astigmia, both by means of its feathers, and by the two barbs of the arrow head. Some eyes are more sensitive in comparing groups of lines than single lines while other eyes too rapidly experience chromatic sensations from the former to make them of service for testing. To use the test the patient need only wear the spherical lens (arrived at by retinoscopy or otherwise) which makes one meridian emmetropic and the remainder myopic, in other words the highest plus (or weakest minus) lens compatible with best vision of the test types. Then the inverted fan will show approximately where to place the arrow to start with. Its point should touch the blackest spoke of the fan, or occupy the middle of the blackest group. If the brightest spokes are scattered in different parts of the fan the astigmatism is "irregular," and the case more suitable for the test type only. The arrow should then be turned in the direction of the clearest feather, or the faintest barb, till the two sides agree in clarity.

D. F. H.

Blatt, Nicolas. Cyclodialysis. Ann. di Ottal. e Clin. Ocul. v. 51, 1923, p. 819.

Thirty-eight cases of simple glaucoma were operated by cyclodialysis. Many of these patients were carefully observed over a period of two years, and none less than one year. In no case did the author note any of the disadvantages referred to in the literature as accompanying the trephining methods, such as postoperative cataract, late infection, iridocyclitis, retinal hemorrhages, etc. The technic employed was that described by Salus in June, 1920, with the difference that Blatt, while withdrawing the spatula imparted to this instrument fairly wide lateral movements in the supraciliary and suprachoroidal space, in order to divide as many ciliary nerves as possible. The complications which supervened in the course of the operation, such as hemorrage into the anterior chamber, iridodialysis, pearling of the vitreous body in the scleral wound, were mostly harmless and little dangerous incidents, which rapidly passed off. The end results at the termination of the period of observation were, as regards acuity of vision, as follows: improved 22 cases, unchanged 19, worse 9; as regards field of vision: improved 16, unchanged 26, worse 8. The cases which presented a very defective field of vision before operation, as well as those with marked hemeralopia, became rapidly worse with respect to visual acuity after operation.

Blatt considers cyclodialysis indicated in all those cases of simple glaucoma in which diminution of the visual field or progressive loss of sight, cannot be prevented with miotics. In order to elucidate the problem of the way in which cyclodialysis accomplishes the reduction of tension, we must consider the neurogenic view of the glaucoma question. Ciliary ganglion, ciliary nerves, the ciliary plexus in the ciliary body and the nerve endings which Seidel has shown to exist between the cells of the ciliary epithelium form the terminals of the central regulating apparatus which transmits to the ciliary body a constant stimulus which allows the intraocular secretion to keep step with the conditions of intraocular pressure. Possibly the tension reducing effect of cyclodialysis is brought about through destruction and solution of continuity of the ciliary nerves of the suprachoroidal and supraciliary tissue. The unknown general factor, whether it be of nervous origin or connected with the internal secretions, persists of course, but is no longer in a position to exercise its harmful local action on the eye when a break has once been made at some point in its path.

St. John.

Hamburger, C. New Treatment of Glaucoma. Klin. M. f. Augenh., 1924, v. 72, p. 47.

Glaucoma, as far as it is due to paresis of the sympathetic nerve (the vasoconstrictors), i. e., to a uveal "flood," is favorably influenced: (1) By subconjunctival injection of epinephrin because this drug stimulates the sympathetic and restores the vascular tonus and thus empties the choroid, which acts with the iris and ciliary body as a large water cushion. (2) By subsequent instillation of eserin, because miotics drive the blood within the eye forward, unfold the iris and increase the absorbing surface at a time when the adrenalin mydriasis recedes. Thus, also, the therapeutic effects of operations are explained. They do not produce fistulization but hyperemia and congestion of the anterior segment, whereby the large posterior water cushion is automatically released.

Hence, the following indications: If the miotics fail, epinephrin injections are to be resorted to, and if the combination of epinephrin and miotics fail, operation is indicated.

C. Z.

Preziosi, C. The Electrocautery in the Treatment of Glaucoma. Brit. J. Ophth., v. 8, 1924, p. 414.

The author summarizes some of the salient points in dealing with simple glaucoma. He refers to de Wecker's having grasped the importance of the filtration of aqueous under the conjunctiva. To this end the Lagrange and subsequently the Elliot trephining operations were devised. The contributor conceived the idea of doing away with the cutting instruments and establishing a fistulous drainage between the anterior chamber and the subconjunctival tissue by means of the electrocautery. A conjunctival flap is laid over the cornea. The cornea is split in two layers for 1.5 mm. With an electrocautery, heated to redness and directed toward the filtration angle, the tissues are touched lightly several times until the aqueous starts flowing. The conjunctival flap is replaced. The cautery is of platinum wire 1 mm. in diameter at the point.

The author claims the following advantages. Simplicity of technic; impossibility of injuring deep structures in those cases where the base of the iris is extensively attached to the posterior surface of the cornea; ease of perforation of anterior chamber; slow emptying of the anterior chamber; absolute asepsis. Iridectomy is not performed so fragments of iris cannot remain entangled in the sclerocorneal aperture. Notes of three patients operated upon are given.

D. F. H.

Rochat, G. F. and Mulder, W. Progressive Atrophy of Iris with Glaucoma. Brit. J. Ophth., v. 8, Aug. 1924, p. 362.

Knowledge concerning this disease is quite meager because of its infrequency, slow course, absence of pain and the rarity of an opportunity for microscopic study. In such studies as have been made, no signs of inflammation were observed. A division of opinion exists as to whether atrophy of the iris is primary or secondary to a slowly progressive glaucoma. The eccentricity of the pupil may be the result of a circumscribed shrinking of the iris but is perhaps just as well explained by assuming a primary atrophy of the iris, the pupil being drawn to the side where the iris is less atrophic, simply as the result of a disturbance of the equilibrium. The authors report a case which they were able to observe from the beginning. The eyes of a woman, aged 41 years, were normal in every way except for a displacement of the pupil out and a rarefaction and vertical eliptical hole in the iris stroma on the nasal side. The eye was again observed six years later at which time it had become blind. There was a + 2 tension and a deep excavation of the disc. The entire nasal half of the iris was occupied by a large hole. The eye was enucleated and submitted to a microscopic examination. The eye was functionally normal at the time of first observation when there already existed a marked degree of iris atrophy. It existed before a trace of glaucoma could be detected. Secondly, the case showed clearly that the displacement

of the pupil could not have been the effect of a primary atrophy of some adjacent part of the iris, for tho at the time of the first examination the hole in the iris was not very large, the pupil was already markedly pulled to one side. From the pathologic examination, as well as the clinical data, one gets the impression that the principal factor in the process was the soldering together of the root of the iris with the periphery of the cornea, beginning at a circumscribed spot and leading to displacement of the pupil. Eight references and three illustrations accompany the contribution.

D. F. H.

Jess, A. Glaucoma in Chalcosis of the Eye. Klin. M. f. Augenh., 1924, v. 72, p. 128.

Within the last five and one-half years, Jess observed thirteen cases of chalcosis (deposit of copper in the tissues) of the eye after injuries by pieces of brass. From his experience, he was inclined to consider the prognosis of slow chalcosis as favorable and, on account of the difficult extraction of the generally minimal chips from the vitreous and fundus, abstained from operative procedures. This conservative standpoint may, however, have to be abandoned, since in two cases the eyes were lost from purulent iridocyclitis five years after the injury. The clinical history of a man, aged 49, is related whose left eye was injured in 1916 by the explosion of a hand grenade, and a piece of brass was lodged behind the lens. After a few years the eye became blind without causing pain or inflammation. When seen by Jess, it showed intense, internal chalcosis and absolute glaucoma.

C. Z.

Jess, A. Aluminum in the Eye. Klin. M. f. Augenh., 1924, v. 72, p. 133.

A man, aged 40, sustained an injury of both eyes by the explosion of a porous aluminum shell. One eye recovered entirely but the other showed a large piece of aluminum in the iris and numerous smaller particles, on the iris and Descemet's membrane, of a luster of silver. The foreign bodies were well tolerated and, excepting slight depigmentation of the pupillary margin, there were no changes after two years. V. = 5/5. The case showed the relatively small danger of injuries by aluminum. Jess corroborated this in experiments on animals.

C. Z.

Kassner, H. Ocular Complications as Late Sequels of Encephalitis Lethargica. Klin. M. f. Augenh., v. 72. p. 59, 1924.

From Jan. 1, 1921, to the end of February, 1923, fifty cases of postencephalitis with eye symptoms were observed at the clinic. The following abnormalities were noted: 1. Disturbance of motility and paresis of the exterior ocular muscles, associated and dissociated paralyses of fixation; ptosis; facial paresis. 2. Sluggish pupillary reaction; irregular pupils; anisocoria; paresis of accommodation. (The tests were made with the pupilloscope of von Hess.) 3. Affections of the fundus.

In most cases, two or more symptoms were found. The ocular complications, which were observed four years after the encephalitis, mainly concerned the exterior and interior muscles. The optic nerve was rarely affected. In no case was the vision changed by the encephalitis. Much similarity was observed in the final stage. These cases mostly show typical, irreparable, anatomic changes, chiefly in the nuclei of oculomotor and abducens in the pons and floor of the Sylvian aqueduct.

C. Z.

Chambers, E. R. Paralysis of Divergence in Encephalitis Lethargica. Brit. J. Ophth, v. 8, 1924, p. 417.

A girl, aged 19 years, following certain head symptoms complained of a diplopia when looking at distant objects. It was hardly present in near vision. The diplopia at three meters, in the six cardinal directions of gaze showed it to be homonymous with erect images, the unusual feature being that there was no alternation in the relative position of the two images in any

of the six cardinal directions of gaze. A second examination carried out at five meters, showed the two images to be further apart than they were when the examination was performed at three meters from the patient, but again, their relative positions remained the same in the six cardinal directions of gaze. On approximating the candle to two feet from the patient, the diplopia ceased to exist in all six cardinal directions of gaze. This case could not have been one of paresis of any individual muscle as there was no increase in the distance between the images in the direction of action of any muscle, nor could spasm of accommodation produce the above type of diplopia, as in this case the double vision would increase as the object was brought nearer the patient and diminish the further it was removed. The condition appears to be due to paralysis of divergence.

D. F. H.

Hepburn, L. Classification of Diseases of the Choroid. Brit. J. Ophth., v. 8, 1924, p. 401.

The author submits a new nomenclature for diseases of the choroid in place of the older and more obsolete classification now in vogue. Instead of the present descriptive regional method, e. g., macular diseases, choroiditis juxtapapillaris, etc., the author bases his classification on the admirable pathologic and clinical work which has been carried on during the past twenty years. They fall into five groups; inflammatory; vascular; degenerative; congenital; new growths.

1. Where there is a large or small edematous patch accompanied by sudden lowering of the visual acuity, vitreous opacities and keratic precipitates, followed by the formation of fibrous tissues of varying amount with many coarse masses of gross pigmentation and sometimes vessels crossing the floor of the scar, the pathologic condition producing it is inflammatory in origin.

2. Where the pigmentary changes are of a fine granular type and the choroidal disturbance, tho decided, is of a somewhat indefinite character, accompanied by variable visual acuity with no vitreous opacities and no keratic precipitates, the pathologic condition producing it is of vascular origin, either complete or partial cutting off of the blood supply, an old hemorrage, etc.

3. When we find white or whitish yellow areas, never very large and sometimes quite minute, surrounded by a perfectly even, well defined border of pigment which is not excessive, with no choroidal disturbance around it, the condition is one of hyalin degeneration probably of the membrane of Bruch.

4. When there is a pearly white patch with absence of vessels crossing the floor of it, and a narrow fringe of pigment round a well defined margin with no choroidal disturbance beyond the actual defect, the probability is that the condition is congenital in origin.

5. The characteristic features of a new growth are: the raised choroidal swelling, often with a well defined non-edematous border, and whatever pigment there is, is indefinitely mixed up with the main mass; there is frequently an ordinary detachment, namely, separation between the neuroepithelial layer and the pigment epithelial layer, somewhat in the neighborhood of the growth, and, if the case can be safely watched, a gradual increase in the size of the swelling is noticed. The presence of new vessels helps the diagnosis. Four illustrations accompany the contribution.

D. F. H.

NEWS ITEMS

Personals and items of interest should be sent to Dr. Melville Black, 424 Metropolitan Building, Denver, Colorado. They should be sent in by the 25th of the month. The following gentlemen have consented to supply news from their respective sections: Dr. Edmond E. Blaauw, Buffalo; Dr. H. Alexander Brown, San Francisco; Dr. V. A. Chapman, Milwaukee; Dr. Robert Fagin, Memphis; Dr. M. Feingold, New Orleans; Dr. Wm. F. Hardy, St. Louis; Dr. Geo. F. Keiper, LaFayette, Indiana; Dr. George H. Kress, Los Angeles; Dr. W. H. Lowell, Boston; Dr. Pacheco Luna, Guatemala City, Central America; Dr. Wm. R. Murray, Minneapolis; Dr. G. Oram Ring, Philadelphia; Dr. Chas. P. Small, Chicago; Dr. John E. Virden, New York City; Dr. John O. McReynolds, Dallas, Texas; Dr. Edward F. Parker, Charleston, S. C.; Dr. Joseph L. McCool, Portland, Oregon; Dr. Richard C. Smith, Superior, Wis.; Dr. J. W. Kimberlin, Kansas City, Mo.; Dr. G. McD. Van Poole, Honolulu; Dr. E. B. Cayce, Nashville, Tenn.; Dr. Gaylord C. Hall, Louisville, Ky.; Dr. Edward D. LeCompte, Salt Lake City.

DEATHS.

Dr. Louis F. Love, Philadelphia, age 63, died November 3, of heart disease.

The death of Dr. Jean Darrieux, of Paris, was announced in the Annales d'Oculistique of October, 1924.

Dr. John B. Roberts of Philadelphia died November 28, 1924, from injuries received when knocked down by a truck. He was best known as a general surgeon but also did much ophthalmic surgery especially plastic operations on the lids.

LONDON CONVENTION, JULY, 14-17.

A preliminary bulletin of the Convention of English Speaking Ophthalmological Societies, July, 1925, will shortly be issued to the members of such societies and associations. It will contain a detachable slip which those wishing to become members of the convention are asked to fill in and send to the secretary of the Finance and Registration Committee, Mr. Leslie Paton, 29 Harley Street, London, W., as soon as possible.

The registration fee for those who are not members of the Ophthalmological Society of the United Kingdom will be 2 pounds, and for ladies accompanying members of the convention who wish to become associate members, 10 shillings. Each member of the convention will receive a copy of its transactions when published.

The following selected speakers will take part in a symposium "On the evolution of binocular vision." Sir Charles Sherrington, F.R.S.; Sir Arthur Keith, F.R.S.; Sir Fredrick Mott, F.R.S.; Prof. Elliot Smith, F.R.S.; Prof. S. E. Whitnall (Montreal).

The discussion on "Microscopy of the living eye," will be opened by Dr. Gordon Byers (Montreal); Mr. Harrison Butler (Birmingham); Dr. Arthur J. Bedell (Albany); and Mr. Basil Graves (London).

Those wishing to take part in this discussion, to read papers, to give demonstrations, or to contribute exhibits to the museum, are requested to communicate with the secretary of the Scientific Business Committee, Mr. R. Foster Moore, 91 Harley Street, London, W.

On the evening of Monday, July 13th, before the opening of the convention, a reception will be held at the Royal College of Surgeons of England. On Saturday, July 18th, at the conclusion of the convention, there will be excursions to Oxford and Cambridge.

SOCIETIES.

Following the October meeting of the Ophthalmological Section of the College of Physicians of Philadelphia, the chairman, Dr. Thomas B. Holloway, invited the members of the section to meet the guest of honor of the evening, Dr. Harry Friedenwald of Baltimore, at the Rittenhouse Hotel where the members had a very delightful social hour together.

The Central Wisconsin Ophthalmic and Otolaryngologic Clinic was organized at Marshfield, October 16th, electing Dr. William Hipke, Marshfield, president; Dr. Arthur L. Payne, Eau Claire, vice-president; and Dr. William G. Merrill, Wisconsin Rapids, secretary-treasurer. The district takes in cities as far north as Ashland and as far south as Fond du Lac. Meetings will be held semi-annually in May and October.

The following papers were read before the Ophthalmological Section of the College of Physicians of Philadelphia at its November meeting: Dr. Louis Lehrfeld, by invitation, "Allergic Reactions in Vernal Conjunctivitis"; Dr. Francis Heed Adler, by invitation, "Demonstration of Normal and Abnormal Pupillary Reflexes"; Dr. Sidney L. Olsho, by invitation, "A Test Chart Screen and a Familiar Test Chart with Letters Reorientated"; Dr. G. E. deSchweinitz; (1) "Factors in the Development of Tobacco-Alcohol Amblyopia." (2) "Senile Changes in the Optic Nerve, the So-Called Senile Amblyopia." (3) "Ocular Disorders Probably Due to Prostatic Infection of Nongonorrheal Origin."

GRADUATE COURSES.

The Chicago Post Graduate Hospital and Medical School announces a special course on the slit lamp microscopy of the living eye with demonstration of use of red free light and the Gullstrand binocular ophthalmoscope, for the week beginning January 12, 1925. The course will consist of illustrated lectures and personal instruction by Dr. Robert Von der Heydt. The class will be limited by the apparatus available. Applications should be made to 2400 South Dearborn Street, Chicago, Illinois.

The University of Pennsylvania announces that arrangements have been effected whereby under the auspices of the Graduate School of Medicine of the University of Pennsylvania, Basil Graves, C.M., of London, England, will

give at the school six identical personal courses in Ocular Microscopy with Slitlamp Illumination, as follows: 1925—first group, January 5th to 17th; second group, January 19th to 31st; third group, February 2nd to 14th; fourth group, February 16th to 28th; fifth group, March 2nd to 14th; sixth group, March 16th to 28th.

Each course will be limited to a group of six ophthalmologists; and will be conducted on five days weekly for two weeks—one hour lecture and one and one-half hours' practice daily. The fee for each course is $100—registration fee of $25 with application for a course; and $75 balance at time of taking the course.

Applications should be sent to Dr. George H. Meeker, dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia. Applications will be honored in the order in which they are received and within the stated group registration limits. More than one selection from the stated course dates should be indicated. If group quotas for requested dates are full registration fee checks will be returned.

A course upon the use of the slit lamp has been given at the Oxford Eye Hospital on December 19, 20, 22 and 23. The course, which was under the general direction of Mr. P. Adams, reader in ophthalmology at Oxford University, was modelled upon that given by Professor Vogt at Zürich. The lectures will be given by Mr. Harrison Butler. Owing to the kindness of Messrs. Zeiss, eight slit lamps will be at the disposal of the hospital. Dr. Franceschetti, first assistant to Professor Vogt, has accepted an invitation to act as demonstrator. The other demonstrators will all be Zürich trained. The number of students is limited to twenty-seven. The fee will be adjusted to pay the expenses of the course, but it is not anticipated that it will exceed 4 pounds.

PERSONAL.

Dr. H. H. McGuire of Winchester, Virginia, has been made president-elect of the Medical Society of Virginia.

Dr. Warren S. Reese has been appointed assistant surgeon at Wills' Eye Hospital, Philadelphia, on the service of Dr. L. F. Appleman.

Dr. William D. Rowland and Dr. Jos. E. Sternberg have been appointed assistant professors in clinical ophthalmology in the Boston University School of Medicine.

Dr. L. V. Stegman of Battle Creek, Michigan, expects to go to India this winter to attend the eye clinic at Shikapur in January.

Dr. William H. Stokes has been promoted from assistant to instructor in ophthalmology, and Dr. Ralph O. Rychener to assistant instructor in ophthalmology, University of Michigan Medical School, Ann Arbor.

Monday evening, November 17th, Dr. Frank Allport of Chicago by invitation read a paper before the Ophthalmological Section of the New York Academy of Medicine, entitled, "Industrial Eye Accidents."

Dr. Lewis H. Taylor announces that Dr. Lewis T. Buckman is now associated with him in practice of Ophthalmology and Otolaryngology at 83 South Franklin Street, Wilkes Barre, Pa.

Dr. Frederick A. Davis, Dr. Eugene E. Neff and Dr. Richard L. Bower announce the formation of a new partnership. Offices will be maintained in the present clinic building to be known hereafter as the Professional Building, 122 West Washington Avenue, Madison, Wisconsin.

Dr. James Moores Ball, of St. Louis, has given his collection of enucleated eyes, pictorial items, microscopic specimens, ophthalmic armamentarium and rare ophthalmic literature to the Army Medical Museum at Washington. A request is made that this collection be added to by the oculists of this country; and that enucleated eyes be preserved in Kaiserling's solution (solution of formaldehyd, 750; distilled water, 1,000; potassium nitrate, 10; potassium acetate, 30), and sent to the curator, Army Medical Museum, Seventh and B Streets, S. W., Washington, D. C.

We note with pleasure the appointments of our English confreres as follows: Mr. O. Gayer Morgan, to assistant ophthalmic surgeon to Guy's Hospital; Mr. F. A. Williamson-Noble, assistant ophthalmic surgeon to St. Mary's Hospital; Dr. Ernest Allan, ophthalmic surgeon to the Stanley Hospital, Liverpool; Dr. G. P. D. Hawker, consulting ophthalmic surgeon to the Devon Mental Hospital, Axminster; Mr. Basil Graves, clinical lecturer and special instructor in "Microscopy of the Living Eye" to the Graduate School of Medicine, University of Pennsylvania for a period of three months.

On October 22nd, Ambassador Jusserand presented the decoration of Commander of the Legion of Honor to Dr. William Howard Wilmer, of Washington, in behalf of the French government. The Washington Post remarks editorially that this rare honor has never been more worthily bestowed. The services Dr. Wilmer performed in France in behalf of the allied soldiers were of priceless value, not merely in the alleviation of suffering and the prevention of blindness, but in installing scientific methods for testing the eyestrain of aviators and the elimination of men who by reason of defective eyesight might have destroyed themselves and others.

Dr. Henry T. Holland, F.R.C.S., E., of Shikapur, India, is enjoying a month's visit in this country. Dr. Holland began his missionary work with the Church of England, at the Quetta Mission, twenty-eight years ago. In 1911 he went to Shikapur and established what has become the largest eye clinic in the world. Because of climatic conditions the clinic is opened only from January 1st to February 15th each year, but in this short period thousands of cases are seen and treated. Dr. Holland with Dr. W. A. Fisher presented a paper at the November meeting of the Chicago Ophthalmogolical Society on "Cataract, with Deductions Drawn from the Observation and Treatment of 1,438 Cases."

Current Literature

These are the titles of papers bearing on ophthalmology. They are given in English, some modified to indicate more clearly their subjects. They are grouped under appropriate heads, and in each group arranged alphabetically, usually by the author's name in **heavy-faced type.** The abbreviations mean: (Ill.) illustrated; (Pl.) plates; (Col. Pl.) colored plates. Abst. shows it is in an abstract of the original article. (Bibl.) mean bibliography and (Dis.) discussion published with a paper.

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Morgan, O. G. Microscopy of living eye. Brit. Med. Jour., 1924, Oct. 25, p. 761.

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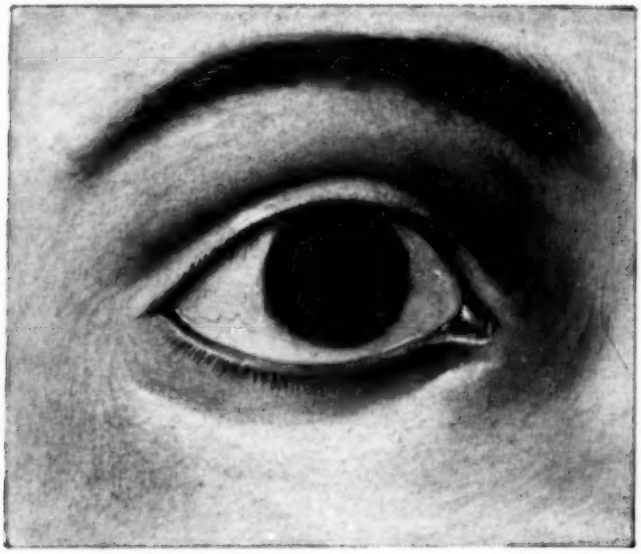

FIG. 1—ZONULAR OPACITY OF CORNEA AS SEEN WITH ZEISS BINOCULAR LOUPE. (KEY'S CASE).

FIG 2—SCHEMATIC REPRESENTATION OF ZONULAR OPACITY UNDER GREATER MAGNIFICATION.

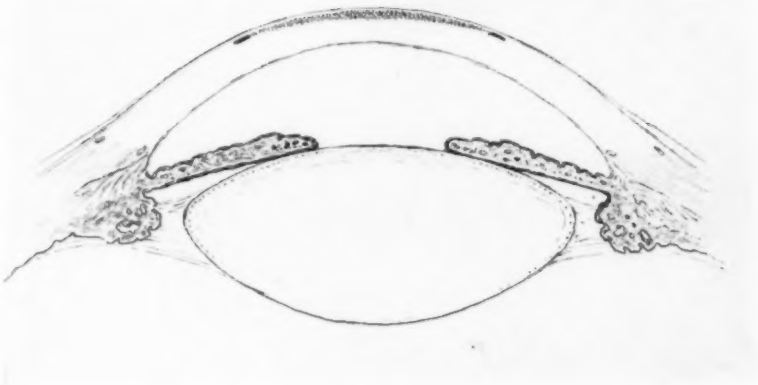

FIG. 3—SCHEMATIC CROSS SECTION OF EYE SHOWING LOCATION OF CORNEAL OPACITY IN KEY'S CASE

FIG. 4—CELL-LIKE BODIES UNDER SLIT LAMP FOCAL ILLUMINATION. OC. 4, OBJ. A 2; SHOWING CHANGE OF COLOR BY STRONGER ILLUMINATION.